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RESPIRATORY CARE

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48th International Respiratory Congress
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October 5-8, 2002, Tampa, Florida

ORIGINAL CONTRIBUTIONS

Air Leak Test and Corticosteroids in Intubated Children

Imposed Work of Breathing During Ventilator Failure

Noninvasive Nocturnal Ventilatory Support in Cystic Fibrosis

Clinical Validation of a Continuous Intravascular Neonatal Blood Gas Sensor

CASE REPORTS

Independent Lung Ventilation in Complex Congenital Heart Disease

Unilateral Expiratory Airflow Obstruction During Forced Exhalation

GUIDELINES, RECOMMENDATIONS, & STATEMENTS

AARC Clinical Practice Guidelines—2002 Revisions & Updates

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- Oxygen Therapy for Adults in the Acute Care Facility
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BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS AND USAGE

DuoNeb is indicated for the treatment of bronchospasm associated with COPD in patients requiring more than one bronchodilator.

CONTRAINDICATIONS

DuoNeb is contraindicated in patients with a history of hypersensitivity to any of its components, or to atropine and its derivatives

WARNINGS

Paradoxical Bronchospasm: In the clinical study of DuoNeb, paradoxical bronchospasm was not observed. However, paradoxical bronchospasm has been observed with both inhaled ipratropium bromide and albuterol products and can be life-threatening. If this occurs, DuoNeb should be discontinued immediately and alternative therapy instituted.

Do Not Exceed Recommended Dose: Fatalities have been reported in association with excessive use of inhaled products containing sympathomimetic amines and with the home use of nebulizers.

Cardiovascular Effect: DuoNeb, like other beta adrenergic agonists, can produce a clinically significant cardiovascular effect in some patients as measured by pulse rate, blood pressure, and/or symptoms. Although such effects are uncommon for DuoNeb at recommended doses, if they occur, the drug may need to be discontinued. In addition, beta agonists have been reported to produce ECG changes, such as flattening of the T-wave, prolongation of the QTc interval, and ST segment depression. The clinical significance of these findings is unknown. Therefore, DuoNeb, like other sympathomimetic amines, should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension.

Immediate Hypersensitivity Reactions: Immediate hypersensitivity reactions to albuterol and/or ipratropium bromide may occur after the administration of DuoNeb as demonstrated by rare cases of urticaria, angioedema, rash, pruritus, oropharyngeal edema, bronchospasm, and anaphylaxis.

PRECAUTIONS

General

1. Effects Seen with Sympathomimetic Drugs: As with all products containing sympathomimetic amines, DuoNeb should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension; in patients with convulsive disorders, hyperthyroidism, or diabetes mellitus, and in patients who are unusually responsive to sympathomimetic amines. Large doses of intravenous albuterol have been reported to aggravate pre-existing diabetes mellitus and ketoacidosis. Additionally, β-agonists may cause a decrease in serum potassium in some patients, possibly through intracellular shunting. The decrease is usually transient, not requiring supplementation.

2. Effects Seen with Anticholinergic Drugs: Due to the presence of ipratropium bromide in DuoNeb, it should be used with caution in patients with narrow-angle glaucoma, prostatic hypertrophy, or bladder-neck obstruction.

3. Use in Hepatic or Renal Diseases: DuoNeb has not been studied in patients with hepatic or renal insufficiency. It should be used with caution in these patient populations.

Information for Patients

The action of DuoNeb should last up to 5 hours. DuoNeb should not be used more frequently than recommended. Patients should be instructed not to increase the dose or frequency of DuoNeb without consulting their healthcare provider. If symptoms worsen, patients should be instructed to seek medical consultation.

Patients must avoid exposing their eyes to this product as temporary papillary dilation, blurred vision, eye pain, or precipitation or worsening of narrow-angle glaucoma may occur, and therefore proper nebulizer technique should be assured, particularly if a mask is used.

If a patient becomes pregnant or begins nursing while on DuoNeb, they should contact their healthcare provider about use of DuoNeb.

See the illustrated Patient's Instruction for Use in the product package insert.

Drug Interactions

Anticholinergic agents: Although ipratropium bromide is minimally absorbed into the systemic circulation, there is some potential for an additive interaction with concomitantly used anticholinergic medications. Caution is, therefore, advised in the co-administration of DuoNeb with other drugs having anticholinergic properties.

β-adrenergic agents: Caution is advised in the co-administration of DuoNeb and other sympathomimetic agents due to the increased risk of adverse cardiovascular effects.

β-receptor blocking agents: These agents and albuterol sulfate inhibit the effect of each other. β-receptor blocking agents should be used with caution in patients with hyperreactive airways, and if used, relatively selective β_1 selective agents are recommended.

Diuretics: The electrocardiogram (ECG) changes and/or hypokalemia that may result from the administration of non-potassium sparing diuretics (such as loop or thiazide diuretics) can be acutely worsened by β-agonists, especially when the recommended dose of the β-agonist is exceeded. Although the clinical significance of these effects is not known, caution is advised in the co-administration of β-agonist-containing drugs, such as DuoNeb, with non-potassium sparing diuretics.

Monoamine oxidase inhibitors or tricyclic antidepressants: DuoNeb should be administered with extreme caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants, or within 2 weeks of discontinuation of such agents because the action of albuterol sulfate on the cardiovascular system may be potentiated.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Albuterol Sulfate. In a 2-year study in Sprague-Dawley rats, albuterol sulfate caused a significant dose-related increase in the incidence of benign leiomyomas of the mesovarium at and above dietary doses of 2 mg/kg (approximately equal to the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). In another study, this effect was blocked by the coadministration of propranolol, a non-selective beta-adrenergic antagonist.

In an 18-month study in CD-1 mice, albuterol sulfate showed no evidence of tumorigenicity at dietary doses up to 500 mg/kg (approximately 140 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). In a 22-month study in Golden hamsters, albuterol sulfate showed no evidence of tumorigenicity at dietary doses up to 50 mg/kg (approximately 20 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis).

Albuterol sulfate was not mutagenic in the Ames test or a mutation test in yeast. Albuterol sulfate was not clastogenic in a human peripheral lymphocyte assay or in an AH1 strain mouse micronucleous assay.

Reproduction studies in rats demonstrated no evidence of impaired fertility at oral doses of albuterol sulfate up to 50 mg/kg (approximately 25 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis).

Ipratropium bromide. In 2-year studies in Sprague-Dawley rats and CD-1 mice, ipratropium bromide showed no evidence of tumorigenicity at oral doses up to 6 mg/kg (approximately 15 times and 8 times the maximum recommended daily inhalation dose for adults in rats and mice respectively, on a mg/m^2 basis).

Ipratropium bromide was not mutagenic in the Ames test and mouse dominant lethal test. Ipratropium bromide was not clastogenic in a mouse micronucleous assay.

A reproduction study in rats demonstrated decreased conception and increased resorptions when ipratropium bromide was administered orally at a dose of 90 mg/kg (approximately 240 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). These effects were not seen with a dose of 50 mg/kg (approximately 140 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis).

Pregnancy

TERATOGENIC EFFECTS: Pregnancy Category C

Albuterol sulfate. *Pregnancy Category C.* Albuterol sulfate has been shown to be teratogenic in mice. A study in CD-1 mice given albuterol sulfate subcutaneously showed cleft palate formation in 5 of 111 (4.5%) fetuses at 0.25 mg/kg (less than the maximum recommended daily inhalation dose for adults on a mg/m^2 basis) and in 10 of 108 (9.3%) fetuses at 2.5 mg/kg (approximately equal to the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). The drug did not induce cleft palate formation when administered subcutaneously at a dose of 0.025 mg/kg (less than the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). Cleft palate formation also occurred in 22 of 72 (30.5%) fetuses from females treated subcutaneously with 2.5 mg/kg isoproterenol (positive control).

A reproduction study in Stride rabbits revealed cranioschisis in 7 of 19 (37%) fetuses when albuterol was administered orally at a dose of 50 mg/kg (approximately 55 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis).

A study in which pregnant rats were dosed with radiolabeled albuterol sulfate demonstrated that drug-related material is transferred from the maternal circulation to the fetus.

During worldwide marketing experience, various congenital anomalies, including cleft palate and limb defects, have been reported in the offspring of patients being treated with albuterol. Some of the mothers were taking multiple medications during their pregnancies. Because no consistent pattern of defects can be discerned, a relationship between albuterol use and congenital anomalies has not been established.

Ipratropium bromide: *Pregnancy Category B.* Reproduction studies in CD-1 mice, Sprague-Dawley rats and New Zealand rabbits demonstrated no evidence of teratogenicity at oral doses up to 10, 100, and 125 mg/kg, respectively (approximately 15, 270, and 680 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). Reproduction studies in rats and rabbits demonstrated no evidence of teratogenicity at inhalation doses up to 1.5 and 1.8 mg/kg, respectively (approximately 4 and 10 times the maximum recommended daily inhalation dose for adults on a mg/m^2 basis). There are no adequate and well-controlled studies of the use of DuoNeb, albuterol sulfate, or ipratropium bromide in pregnant women. DuoNeb should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Labor and Delivery

Oral albuterol sulfate has been shown to delay preterm labor in some reports. Because of the potential of albuterol to interfere with uterine contractility, use of DuoNeb during labor should be restricted to those patients in whom the benefits clearly outweigh the risks.

Nursing Mothers

It is not known whether the components of DuoNeb are excreted in human milk. Although lipid-insoluble quaternary bases pass into breast milk, it is unlikely that ipratropium bromide would reach the infant to an important extent, especially when taken as a nebulized solution. Because of the potential for tumorigenicity shown for albuterol sulfate in some animals, a decision should be made whether to discontinue nursing or discontinue DuoNeb, taking into account the importance of the drug to the mother.

Pediatric Use

The safety and effectiveness of DuoNeb in patients below 18 years of age have not been established.

Geriatric Use

Of the total number of subjects in clinical studies of DuoNeb, 62 percent were 65 and over, while 19 percent were 75 and over. No overall differences in safety or effectiveness were observed between these subjects and younger subjects, and other reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out.

ADVERSE REACTIONS

Adverse reaction information concerning DuoNeb was derived from the 12-week controlled clinical trial.

ADVERSE EVENTS OCCURRING IN ≥ 1% OF ≥ 1 TREATMENT GROUP(5) AND WHERE THE COMBINATION TREATMENT SHOWED THE HIGHEST PERCENTAGE

Body System COSTART Term	Albuterol n (%)	Ipratropium n (%)	DuoNeb n (%)
NUMBER OF PATIENTS	761	754	765
N (%) Patients with AE	327 (43.0)	329 (43.6)	367 (48.0)
BODY AS A WHOLE			
Pain	8 (1.1)	4 (0.5)	10 (1.3)
Pain chest	11 (1.4)	14 (1.9)	20 (2.6)
DIGESTIVE			
Diarrhea	5 (0.7)	9 (1.2)	14 (1.8)
Dyspepsia	7 (0.9)	8 (1.1)	10 (1.3)
Nausea	7 (0.9)	6 (0.8)	11 (1.4)
MUSCULO-SKELETAL			
Cramps leg	8 (1.1)	6 (0.8)	11 (1.4)
RESPIRATORY			
Bronchitis	11 (1.4)	13 (1.7)	13 (1.7)
Lung Disease	36 (4.7)	34 (4.5)	49 (6.4)
Pharyngitis	27 (3.5)	27 (3.6)	34 (4.4)
Pneumonia	7 (0.9)	8 (1.1)	10 (1.3)
UROGENITAL			
Infection urinary tract	3 (0.4)	9 (1.2)	12 (1.6)

Additional adverse reactions reported in more than 1% of patients treated with DuoNeb included constipation and voice alterations.

In the clinical trial, there was a 0.3% incidence of possible allergic-type reactions, including skin rash, pruritus, and urticaria.

Additional information derived from the published literature on the use of albuterol sulfate and ipratropium bromide singly or in combination includes precipitation or worsening of narrow-angle glaucoma, acute eye pain, blurred vision, paradoxical bronchospasm, wheezing, exacerbation of COPD symptoms, drowsiness, aching, flushing, upper respiratory tract infection, palpitations, taste perversion, elevated heart rate, sinusitis, back pain and sore throat.

DOSAGE AND ADMINISTRATION

The recommended dose of DuoNeb is one 3 mL vial administered 4 times per day via nebulization with up to 2 additional 3 mL doses allowed per day, if needed. Safety and efficacy of additional doses or increased frequency of administration of DuoNeb beyond these guidelines has not been studied and the safety and efficacy of extra doses of albuterol sulfate or ipratropium bromide in addition to the recommended doses of DuoNeb have not been studied.

The use of DuoNeb can be continued as medically indicated to control recurring bouts of bronchospasm. If a previously effective regimen fails to provide the usual relief, medical advice should be sought immediately, as this is often a sign of worsening COPD, which would require reassessment of therapy.

A Pari-LC-Plus™ nebulizer (with face mask or mouthpiece) connected to a PRONEB™ compressor was used to deliver DuoNeb to each patient in one U.S. clinical study. The safety and efficacy of DuoNeb delivered by other nebulizers and compressors have not been established.

DuoNeb should be administered via jet nebulizer connected to an air compressor with an adequate air flow, equipped with a mouthpiece or suitable face mask.

HOW SUPPLIED

DuoNeb is supplied as a 3-mL sterile solution for nebulization in sterile low-density polyethylene unit-dose vials. Cards of five vials are placed into a foil pouch. Supplied in cartons as listed below.

NDC 49502-672-30 30 vials per carton
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Store between 2°C and 25°C (36°F and 77°F). Protect from light.

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03-485-21(BRS) May 2001

1. Gross N, Tashkin D, Miller R, et al. Inhalation by nebulization of albuterol-ipratropium combination (Dey combination) is superior to either agent alone in the treatment of chronic obstructive pulmonary disease. *Respiration.* 1998;65:354-362.

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ORIGINAL CONTRIBUTIONS

The Use of the Air Leak Test and Corticosteroids in Intubated Children: A Survey of Pediatric Critical Care Fellowship Directors
by Jason A Foland, Dennis M Super, Nagib S Dahdah, Maroun J Mhanna—Cleveland, Ohio **662**

Imposed Work of Breathing During Ventilator Failure
by Paul N Austin, Robert S Campbell, Jay A Johannigman, and Richard D Branson—Cincinnati, Ohio **667**

Noninvasive Nocturnal Ventilatory Support in Advanced Lung Disease from Cystic Fibrosis
by John T Granton, Colin Shapiro, and Steven Kesten—Toronto, Ontario, Canada **675**

Clinical Validation of a Continuous Intravascular Neonatal Blood Gas Sensor Introduced Through an Umbilical Artery Catheter
by Patricia A Meyers, Catherine Worwa, Robert Trusty, and Mark C Mammel—St Paul, Minnesota **682**

CASE REPORTS

Independent Lung Ventilation in a Patient with Complex Congenital Heart Disease
by Donna Levine, Tina Carmichael, and Peter C Laussen—Boston, Massachusetts **688**

Unilateral Expiratory Airflow Obstruction During Forced Exhalation
by Umur Hatipoglu, Franco Laghi, Steven E Cattapan, Arcot J Chandrasekhar—Maywood, Illinois **693**

LETTERS

Is the Gastrointestinal Tract the Sole Source of Organisms in Ventilator-Associated Pneumonia?
by Robert R Demers—Carmel, California
And Now for the Rest of the Story
response to Demers by Dean R Hess—Boston, Massachusetts **696**

BOOKS, FILMS, TAPES, & SOFTWARE

Bronchial Asthma: Principles of Diagnosis and Treatment, 4th ed. (Gershwin ME, Albertson TE, editors)
reviewed by Mary Hart and Mark W Millard—Dallas, Texas **700**

Manual of Asthma Management, 2nd ed (O'Byrne PM, Thomson NC, editors)
reviewed by Rajeev V Venkayya—San Francisco, California **701**

CRC Desk Reference for Allergy and Asthma (Simon H-U)
reviewed by Melissa K Brown—San Diego, California **702**

Allergies and Asthma for Dummies (Berger WE)
reviewed by Karen J Crogan—Seattle, Washington **703**

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CONTENTS

CONTINUED...

ALSO IN THIS ISSUE

727 AARC Membership Application

651 Abstracts from Other Journals

736 Advertisers Index & Help Lines

736 Author Index

735 Calendar of Events

729 Manuscript Preparation Guide

733 New Products

734 Notices

Control Your Child's Asthma: A Breakthrough Program for the Treatment and Management of Childhood Asthma (Farber HJ, Boyette M) 704
reviewed by James P Kemp—San Diego, California

Environmental Asthma (Bush RK, editor) 705
reviewed by Stephen T Holgate—Southampton, United Kingdom

Cilia and Mucus: From Development to Respiratory Defense (Salathe M, editor) 706
reviewed by Markus O Henke and Bruce K Rubin—Winston-Salem, North Carolina

GUIDELINES, RECOMMENDATIONS, & STATEMENTS

AARC Clinical Practice Guidelines

Selection of an Oxygen Delivery Device for Neonatal and Pediatric Patients—2002 Revision & Update 707

Oxygen Therapy for Adults in the Acute Care Facility—2002 Revision & Update 717

In-Hospital Transport of the Mechanically Ventilated Patient—2002 Revision & Update 721

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Editorials, Commentaries, and Reviews to Note

Psychological Factors in Asthma Control and Attack Risk (editorial)—Osman LM. Thorax 2002 Mar;57(3):190-191.

BYD Guideline: Non-Invasive Ventilation in Acute Respiratory Failure. British Thoracic Society Standards of Care Committee. Thorax 2002 Mar;57(3):192-211.

An Epidemic of Inhalation Anthrax, the First in the Twentieth Century: I. Clinical Features. 1960—Plotkin SA, Brachman PS, Utell M, Bumford FH, Atchison MM. Am J Med 2002 Jan;112(1):4-12; discussion 2-3.

Cost Effectiveness of Continuing Professional Development in Health Care: A Critical Review of the Evidence—Brown CA, Belfield CR, Field SJ. BMJ 2002 Mar 16;324(7338):652-655.

Ventilatory Failures with the Datex-Ohmeda 7900 SmartVent—Cantillo J, Domsky R, Gratz I, Goldberg ME. Anesthesiology 2002 Mar;96(3):766-768.

Oxygen and Resuscitation: Beyond the Myth—Lefkowitz W. Pediatrics 2002 Mar;109(3):517-519.

Accuracy of Information on Apparently Credible Websites: Survey of Five Common Health Topics—Kunst H, Groot D, Latthe PM, Latthe M, Khan KS. BMJ 2002 Mar 9;324(7337):581-582.

How to Find the Good and Avoid the Bad or Ugly: a Short Guide to Tools for Rating Quality of Health Information on the Internet—Wilson P. BMJ 2002 Mar 9;324(7337):598-602.

Sleep-Disordered Breathing, Control of Breathing, Respiratory Muscles, and Pulmonary Function Testing in AJRCCM 2001—Tobin MJ. Am J Respir Crit Care Med 2002 Mar 1;165(5):584-597.

Asthma, Airway Biology, and Nasal Disorders in AJRCCM 2001—Tobin MJ. Am J Respir Crit Care Med 2002 Mar 1;165(5):598-618.

Pediatrics, Surfactant, and Cystic Fibrosis in AJRCCM 2001—Tobin MJ. Am J Respir Crit Care Med 2002 Mar 1;165(5):619-630.

Chronic Obstructive Pulmonary Disease, Pollution, Pulmonary Vascular Disease, Transplantation, Pleural Disease, and Lung Cancer in AJRCCM 2001—Tobin MJ. Am J Respir Crit Care Med 2002 Mar 1;165(5):642-662.

Supplemental Oxygen Use in Ischemic Stroke Patients: Does Utilization Correspond to Need for Oxygen Therapy?—Pancioli AM, Bullard MJ, Grulee ME, Jauch EC, Perkis DF. Arch Intern Med 2002 Jan 14;162(1):49-52.

BACKGROUND: In 1994, the American Heart Association Stroke Council concluded that there were no data to support the routine use of supplemental oxygen in patients who had a stroke. More recently, supplemental oxygen has been suggested to be potentially detrimental. The purpose of this study was to determine the extent of oxygen use in ischemic stroke patients and whether patients receiving oxygen had indications for its use. METHODS: A literature search was performed to generate a comprehensive list of explicit criteria for supplemental oxygen use. When the literature disagreed, the criteria were included in the list to overestimate rather than underestimate the justification for oxygen use. A retrospective chart review of consecutive, nonintubated, ischemic stroke patients admitted to a university hospital was performed. Statistical tests and logistic regression models were constructed to identify the presence of unjustified oxygen use within the sample. Hospital charges were used to quantify opportunities for resource conservation. RESULTS: A total of 167 patient charts were reviewed yielding a total of 600 inpatient days abstracted. One hundred two patients (61.1%) received oxygen during some portion of their hospitalization. Of the 322 days that patients received oxygen, 147 (45.6%) met at least 1 criterion for oxygen use. Of the 278 days that patients did not receive oxygen, 69 (24.8%) met at least 1 of the criteria for oxygen use. There were 384 days for which no criteria were met. Of these, a patient still received oxygen 45.6% of the time (175 days). Factors associated with oxygen use included the presence of at least 1 justifying criteria as well as increasing age and male sex. Withholding oxygen from those not medically justified by the criteria could produce resource savings of roughly 45%. CONCLUSIONS: Using a literature-based list of criteria for supplemental oxygen use, only 45.6% of days of oxygen use were justified in our ischemic stroke population. This study demonstrates that oxygen therapy is commonly given to ischemic stroke patients without clear indication, and opportunities exist for substantial resource conservation.

Mechanical Ventilation with Permissive Hypercapnia Increases Intrapulmonary Shunt in Septic and Nonseptic Patients with Acute Respiratory Distress Syndrome—Pfeiffer B, Hachenberg T, Wendt M, Marshall B. Crit Care Med 2002 Feb;30(2):285-289.

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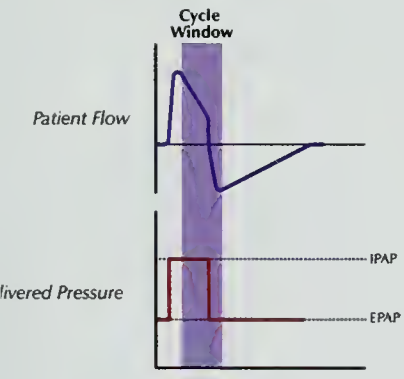

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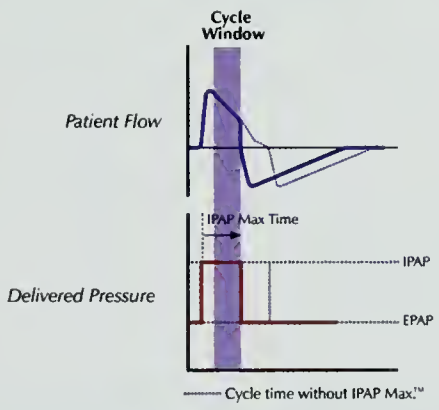

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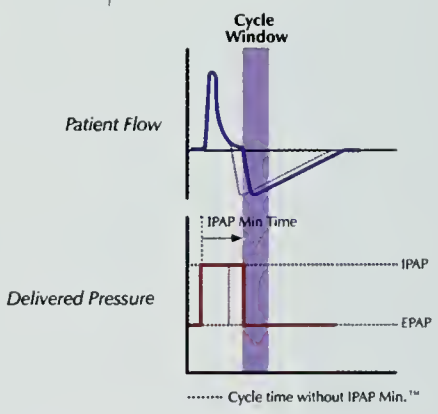

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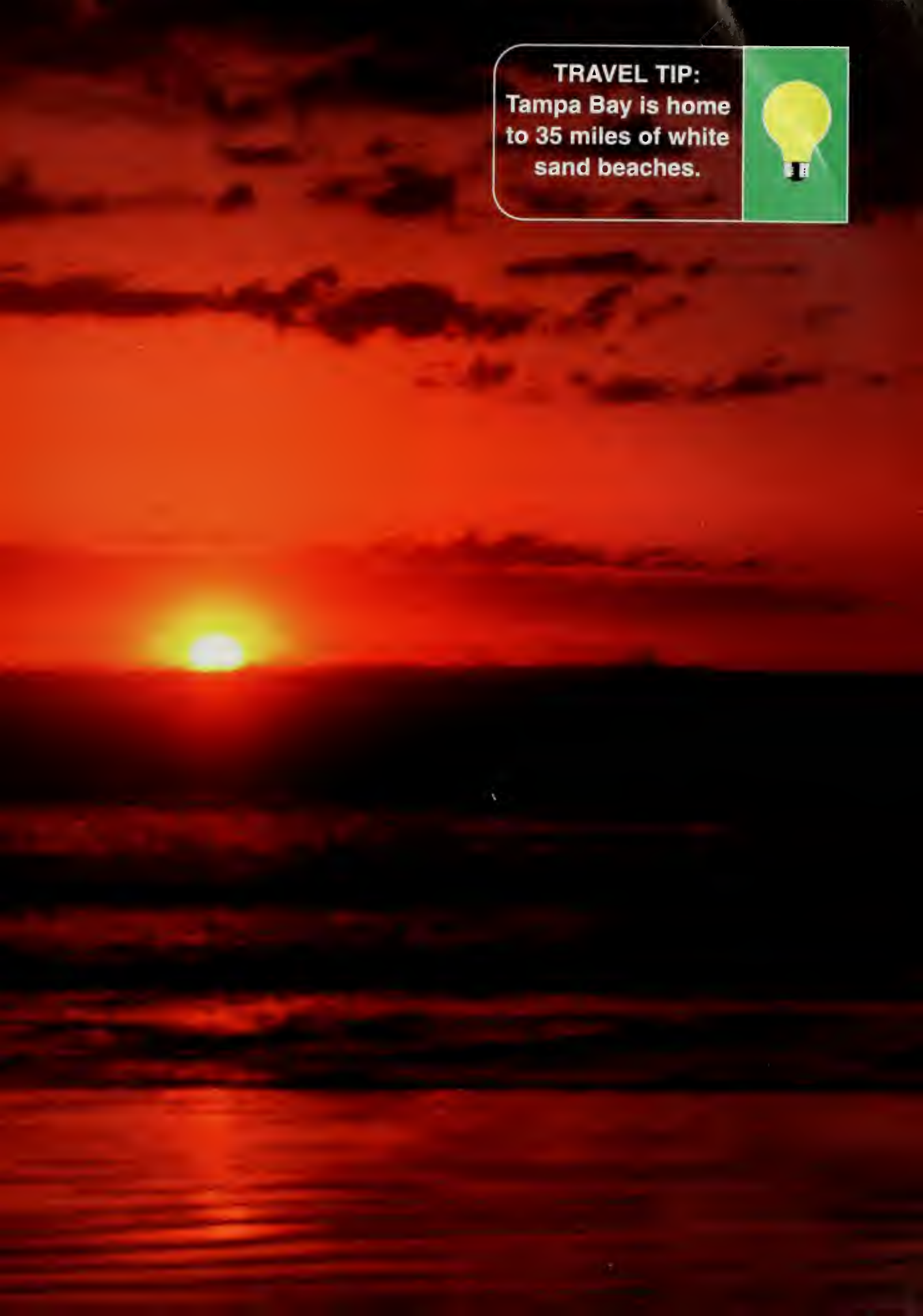
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OBJECTIVE: To compare the effects of conventional mechanical ventilation with low-volume, pressure-limited ventilation (LVPLV) and permissive hypercapnia on ventilation-perfusion ($\dot{V}/\dot{Q}$) distributions in patients with acute respiratory distress syndrome. We hypothesized that the advantageous cardiopulmonary effects of LVPLV would be greater in patients with sepsis than in those without sepsis. PATIENTS AND INTERVENTIONS: Twenty-two patients with acute respiratory distress syndrome were studied (group 1: 12 patients with hyperdynamic sepsis; group 2: 10 nonseptic patients). Intrapulmonary shunt ($\dot{Q}_{sp}/\dot{Q}_t$) (percentage of cardiac output), perfusion of "low" $\dot{V}/\dot{Q}$ areas (percentage of cardiac output), ventilation of "high" $\dot{V}/\dot{Q}$ areas (percentage of total ventilation [$\dot{V}_E$]), and deadspace ventilation (percentage of $\dot{V}_E$) were calculated from the retention/excretion data of six inert gases. Data were obtained during conventional mechanical ventilation and during LVPLV. MEASUREMENTS AND MAIN RESULTS: In group 1, LVPLV increased P_{aCO_2} from 38 ± 6 torr (5.1 ± 0.8 kPa) to 61 ± 12 torr (8.1 ± 1.6 kPa). $\dot{Q}_{sp}/\dot{Q}_t$ increased from 28 ± 16% to 36 ± 17%, whereas P_{aO_2} (84 ± 15 torr [11.1 ± 2.0 kPa] vs. 86 ± 21 torr [11.5 ± 2.8 kPa]) and $\dot{Q}_t$ (10.6 ± 2.3 vs. 11.5 ± 2.5 L x $^{-1}$) remained unchanged and $P_{\bar{v}O_2}$ (40 ± 4 [5.3 ± 0.5 kPa] vs. 49 ± 6 torr [6.5 ± 0.3]) increased. In group 2, LVPLV increased P_{aCO_2} from 38 ± 6 torr (5.1 ± 0.8 kPa) to 63 ± 11 torr (8.4 ± 1.5 kPa). For $\dot{Q}_{sp}/\dot{Q}_t$ (24 ± 9% to 34 ± 16%), the increase was not significant, whereas $\dot{Q}_t$ (7.4 ± 1.8 vs. 10.2 ± 2.2 L x $^{-1}$), $P_{\bar{v}O_2}$ (38 ± 4 torr [5.1 ± 0.5 kPa] vs. 50 ± 6 mm Hg [6.7 ± 0.8 kPa]), and P_{aO_2} (89 ± 16 torr [11.9 ± 2.1 kPa] vs. 98 ± 19 torr [13.1 ± 2.5 kPa]) increased. In both groups, the scatter of perfusion distribution (log SD_Q) was greater than expected for normal subjects but was not different between the groups or altered by the treatments. CONCLUSIONS: In patients with acute respiratory distress syndrome, LVPLV with permissive hypercapnia, tended to increase $\dot{Q}_{sp}/\dot{Q}_t$, without a concomitant decrease of P_{aO_2}. This occurs because, although atelectasis and increased shunt result from the low ventilatory volume, the effects on P_{aO_2} are offset by increased $P_{\bar{v}O_2}$ resulting from the hypercapnic stimulation of cardiac output. This result was independent of the presence or absence of sepsis.

Education, Ethics, and End-of-Life Decisions in the Intensive Care Unit—Stevens L, Cook D, Guyatt G, Griffith L, Walter S, McMullin J. Crit Care Med 2002 Feb;30(2):290-296.

OBJECTIVE: To examine the influence of education and clinical experience on residents' attitudes toward withdrawal of life support. DESIGN: Self-administered survey. SETTING: Four Canadian teaching hospitals. SUBJECTS: Residents rotating through four intensive care units. MEASUREMENTS AND MAIN RESULTS: The survey examined ethics education and experience regarding end-of-life care, importance of factors influencing withdrawal of life support, confidence in decisions, and recommendations for enhancing end-of-life education. The response rate was 83.9% (52 of 62). A minority of residents reported an appropriate amount of formal teaching on ethical principles (17.3%), patient-centered education (28.8%), and informal discussion (28.8%) before their intensive care unit rotation. During their rotation, most residents cared for patients in whom withdrawal of life support was considered. Although they usually attended family meetings, residents were never (34.6%) or rarely (42.3%) the primary discussant. Before the intensive care unit rotation, confidence in withdrawal decisions was related to male sex ($p = 0.001$) and previous patient-centered ethics education ($p = 0.02$). At the end of the intensive care unit rotation, only resident involvement in family meetings ($p = 0.02$) and being the primary discussant at such meetings ($p = 0.01$) were associated with confidence. After we adjusted for prerotation confidence in withdrawal of life support decision-making, the only predictor of postrotation confidence was family meeting involvement ($p < 0.001$). Residents recommended more patient-centered discussion, observation of attending physicians discussing end-of-life issues, and opportunity to lead family meetings. CONCLUSIONS: Experiential, case-based, patient-centered curricula are associated with resident confidence in withdrawal of life support decisions in the intensive care unit.

Brain Death Assessment Using Instant Spectral Analysis of Heart Rate Variability—Baillard C, Vivien B, Mansier P, Mangin L, Jasson S, Riou B, Swynghedauw B. Crit Care Med 2002 Feb;30(2):306-310.

OBJECTIVE: Confirmation of brain death requires an urgent diagnosis to allow rapid vital organ removal for transplantation. Evaluation of forebrain functions is commonly performed through electroencephalogram. Nevertheless, there are, for the moment, no methods that allow for an instantaneous evaluation of brainstem functions. During acute brain injury, heart rate variability is an independent neurologic prognosis indicator resulting from a close relationship between brain stem and cardiac autonomic nervous system. This study aims to evaluate a new heart rate variability spectral analysis method, on a beat-to-beat basis, continuously over the time, during brain death. DESIGN: Prospective, nonrandomized, observational study. SETTING: Intensive care unit. SUBJECTS: Ten patients (age range 25-64 yrs, mean age 41 yrs) with acute brain injury leading to brain death. INTERVENTION: No intervention beyond standard of care MEASUREMENTS AND MAIN RESULTS: Heart rate, arterial blood pressure, heart rate variability in time and frequency domains method, which included calculation of the instant center frequency of spectrum. Brain death was associated with tachycardia (R-R interval 703 ± 69 vs. 551 ± 34 msec, $p < 0.05$), dramatic reduction of the global spectral power (44.919 ± 31.511 vs. 3.204 ± 1.469 msec^2, $p < 0.05$), and an abrupt shift of instant center frequency to a higher frequency range (0.17 ± 0.01 vs. 0.26 ± 0.03 Hz, $p < 0.05$). CONCLUSIONS: Such a method allows an instant, noninvasive determination of brainstem death based on a time and frequency domain analysis of heart rate variability.

Noninvasive Proportional Assist Ventilation Compared with Noninvasive Pressure Support Ventilation in Hypercapnic Acute Respiratory Failure—Wysocki M, Richard JC, Meshaka P. Crit Care Med 2002 Feb;30(2):323-329.

OBJECTIVES: To compare short-term administration of noninvasive proportional assist ventilation (NIV-PAV) and pressure support ventilation (NIV-PSV). DESIGN: Prospective, crossover, randomized study. SETTING: Medicosurgical intensive care unit in a nonteaching hospital. PATIENTS: Twelve chronic obstructive pulmonary disease patients admitted for hypercapnic acute respiratory failure. INTERVENTION: NIV-PSV and NIV-PAV given in a randomized order after baseline evaluation in continuous positive airway pressure. Using a flow-triggering ventilator, NIV-PAV was adjusted using the runaway method and compared with NIV-PSV at similar peak inspiratory airway pressure. MEASUREMENTS AND MAIN RESULTS: Flow, airway pressure, and changes in esophageal pressure were measured and the tidal volume, the patient's inspiratory work of breathing, and the esophageal pressure—time product were calculated. Arterial pH and P_{aCO_2} were measured and breathing comfort was assessed using a visual analogic scale. Peak inspiratory airway pressure (17 ± 3 cm H_2O) and tidal volume were similarly increased with the two modalities with no change in respiratory rate. The change in esophageal pressure was similarly decreased (from 20 ± 8 cm H_2O in continuous positive airway pressure to 12 ± 7 in NIV-PSV and 10 ± 5 cm H_2O in NIV-PAV) as well as inspiratory muscle effort indexes. Arterial pH and P_{aCO_2} were similarly improved. Breathing comfort was significantly improved in NIV-PAV (+38 ± 38%) but not in NIV-PSV (+11 ± 23%). The tidal volume was more variable in NIV-PAV (89 ± 18%) than in NIV-PSV (15 ± 8%) and changes in tidal volume variability were significantly correlated ($p = 0.02$) with changes in breathing comfort. CONCLUSIONS: In chronic obstructive pulmonary disease patients with hypercapnic acute respiratory failure, NIV-PAV was able to unload inspiratory muscles similarly to NIV-PSV but may be more comfortable than NIV-PSV.

Energy Balance in the Intubated Human Airway Is an Indicator of Optimal Gas Conditioning—Ryan SN, Rankin N, Meyer E, Williams R. Crit Care Med 2002 Feb;30(2):355-361.

OBJECTIVE: The optimal level of inspired heat and humidity for patients receiving long-term mechanical ventilation is still the subject of debate. Many laboratory studies examining surrogate markers for optimal humidity suggest that inspired gas should be at body temperature and fully saturated. The aim of this study was to determine the inspired gas condition that was thermodynamically neutral to the airway of intubated patients, and also examine the contribution of the endotracheal tube to airway heat and water balance. DESIGN: Prospective, block-randomized, observational study. SETTING: General adult intensive care unit of a metropolitan teaching hospital. PATIENTS: Ten adult patients requiring intermittent positive pressure ventilation for nonpulmonary reasons. INTERVENTIONS: Each patient was given four different gas conditions—30 ° C, 30 mg/L; 34 ° C, 38 mg/L; 37 ° C, 44 mg/L; and 40 ° C, 50 mg/L—to breathe in random order. MEASUREMENTS AND MAIN RESULTS: Inspired and expired gas temperature and humidity, and the temperature gradient down the endotracheal tube, were measured and the inspired gas condition that gave thermodynamic neutrality was determined. This was found to be gas at body temperature, saturated. Airway workload and airway water loss increased linearly as the inspired gas departed from this condition, at approximately 1.4 kJ/hr/ ° C and 0.5 mL/hr/ ° C, respectively. The endotracheal tube contributed very little to heat and water exchange. CONCLUSIONS: Inspired gas at body temperature and saturated is thermodynamically neutral to the intubated airway, and thus may be considered the optimal condition for ventilation lasting more than a few hours.

Diagnostic and Therapeutic Management of Nosocomial Pneumonia in Surgical Patients: Results of the Eole Study—Montravers P, Veber B, Auboyer C, Dupont H, Gauzit R, Korinek AM, et al. Crit Care Med 2002 Feb;30(2):368-375.

OBJECTIVE: To assess clinical, microbiological, and therapeutic features of nosocomial pneumonias in surgical patients. DESIGN: Prospective (October 1997 through May 1998), consecutive case series analysis of patients suspected of having pneumonia during the fortnight after a surgical procedure or trauma and receiving antibiotic therapy prescribed by the attending physician for this diagnosis. SETTING: A total of 230 study centers in teaching (n = 66) and nonteaching hospitals (n = 164) (surgical wards and intensive care units). PATIENTS: A total of 837 evaluable patients (mean age 61 ± 18 yrs) including 629 intensive care unit patients. INTERVENTION: None. MEASUREMENTS AND MAIN RESULTS: The diagnostic and therapeutic procedures followed were based on guidelines. Antibiotics and any changes of therapy and duration of treatment were decided by the attending physician. The charts were reviewed by a panel of experts that classified the cases according to clinical, radiologic, and microbiological criteria (when available). The efficacy of treatment was evaluated over a 30-day period following the index episode. The patients were classified into three groups: definite pneumonia (n = 261), possible pneumonia (n = 392), or low-probability pneumonia (n = 184). Ventilator-acquired pneumonia was reported in 303 patients. Early onset pneumonia was reported in 512 cases. Microbiological sampling was performed in 718 patients, by bronchoscopy in 367 cases, recovering 450 organisms in 328 patients, including 94 polymicrobial specimens. High proportions of Gram-negative bacteria and staphylococci were cultured, even in early onset pneumonias. Antibiotic therapy was administered for 13 ± 4 days, using monotherapy in 254 cases. Changes in the initial antibiotic therapy (135 monotherapies) were decided in 517 patients (including clinical failure or persistent infection, n = 171; organisms resistant to initial therapy, n = 177; pulmonary superinfection, n = 68). Death occurred in 180 patients, related to pneumonia in 53 cases. CONCLUSIONS: Nosocomial pneumonias in surgical patients are characterized by high frequency of early onset pneumonia, high proportion of nosocomial organisms even in these early onset pneumonias, and moderate mortality rate.

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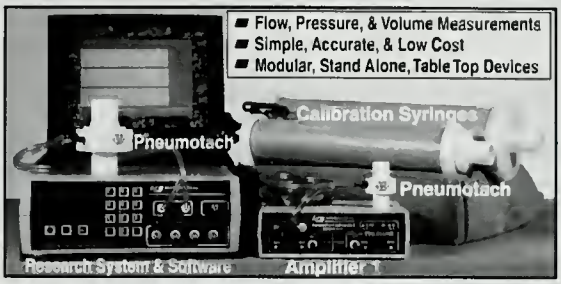

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Beneficial Effects of Nitric Oxide Inhalation on Pulmonary Bacterial Clearance—Jean D, Maitre B, Tankovic J, Meignan M, Adnot S, Brun-Buisson C, et al. Crit Care Med 2002 Feb;30(2):442-447.

OBJECTIVE: The beneficial effects of nitric oxide inhalation on oxygenation during acute respiratory distress syndrome are well described. In contrast, the effects of nitric oxide on pulmonary inflammatory response are much less known in vivo. The objectives of this study were to evaluate the effects of nitric oxide inhalation on bacterial clearance during bacterial pneumonia and on alveolar neutrophil functions. DESIGN: Controlled animal study. SETTING: Research laboratory of an academic institution. SUBJECTS: Male Sprague-Dawley rats. INTERVENTIONS: Severe pneumonia was induced by alveolar instillation of live *Pseudomonas aeruginosa* (1.5×10^8 colony-forming units/kg) in rats. After instillation, rats were exposed to oxygen alone (F_{IO_2} 100%) or to oxygen (F_{IO_2} approximately 100%) plus nitric oxide (10 ppm) during 24 hrs. MEASUREMENTS AND MAIN RESULTS: Oxygen plus nitric oxide inhalation compared with oxygen alone increased recruitment of alveolar neutrophils ($32.5 \pm 4.6 \times 10^6$ cells/mL vs. $23.4 \pm 1.9 \times 10^6$ cells/mL, $p < 0.05$) and improved bacterial clearance in the bronchoalveolar lavage fluid ($8.1 \pm 4.2 \times 10^2$ vs. $1.6 \pm 1.0 \times 10^5$ colony-forming units/mL, $p < 0.05$) and in the pulmonary parenchyma ($1.7 \pm 1.14 \times 10^7$ vs. $3.4 \pm 1.5 \times 10^8$ colony-forming units/mL, $p < 0.05$). However, neither protein concentration in the bronchoalveolar lavage fluid nor mortality rates were modi-

fied by nitric oxide inhalation. The ex vivo alveolar neutrophil functions were similar regardless of whether rats previously inhaled nitric oxide. In vitro experiments demonstrated that nitric oxide donor had a direct bactericidal effect against *P. aeruginosa* and did not modify alveolar neutrophil functions. CONCLUSIONS: These results suggest a beneficial effect of nitric oxide inhalation on bacterial clearance of *P. aeruginosa* attributable to both a direct bactericidal effect and an influx of alveolar neutrophils with preserved functions.

Randomized, Double-Blind, Placebo-Controlled Trial of Intravenous Salbutamol and Nebulized Ipratropium Bromide in Early Management of Severe Acute Asthma in Children Presenting to an Emergency Department—Browne GJ, Trieu L, Van Asperen P. Crit Care Med 2002 Feb;30(2):448-453.

BACKGROUND: In acute severe asthma, treatment must be initiated early to reverse the pathophysiology that may render airways less responsive to bronchodilation. The addition of nebulized ipratropium bromide to initial emergency department therapy improves pulmonary function, but it is unclear whether this approach results in earlier hospital discharge. The early use of bolus intravenous salbutamol has also been shown to improve outcome, including earlier discharge. We therefore assessed the relative benefits of intravenous salbutamol and nebulized ipratropium bromide in the early management of acute severe asthma in children by a double-blind, randomized, controlled trial. METHODS: This study was undertaken at a tertiary children's hospital, The Children's Hospital at Westmead, The Royal Alexandra Hospital for Children, Westmead, Sydney, Australia. Only children with severe acute asthma as determined by the National Asthma Campaign guidelines criteria and pulmonary index were included. All children received initial nebulized salbutamol therapy (2.5-5 mg salbutamol in 4 mL of normal saline depending on age) at initial emergency department presentation. If asthma remained severe 20 mins later, an intravenous cannula was inserted and intravenous methylprednisolone (1 mg/kg) was administered to all children receiving nebulized salbutamol every 20 mins. Children were then randomized to one of three groups: intravenous salbutamol (15 µg/kg as a single bolus over 10 mins), ipratropium bromide (250 µg), or intravenous salbutamol plus ipratropium bromide. All observers were blinded to treatment groups. Children were randomly assigned to receive a single-dose intravenous bolus of either saline or salbutamol and either nebulized saline or ipratropium bromide determined by a number generated randomly in the hospital pharmacy. The primary outcomes were recovery time and discharge time of each group. Respiratory and hemodynamic monitoring were continuous during the first 2 hrs of the study and then children were monitored clinically for 24 hrs. RESULTS: A total of 55 children with acute severe asthma were entered into the study over an 18-month period. The three groups were similar demographically, with a mean age of 5.9 yrs, and mean duration of attack of 19.6 hrs. No side effects or treatment intolerance were reported. Children in the groups that received intravenous salbutamol had a significant reduction in recovery time to achieving second hourly inhaled salbutamol ($p = 0.008$) compared with those administered inhaled bronchodilator alone. The addition of ipratropium bromide to intravenous salbutamol provided no significant further benefit in terms of nebulizer therapy (intravenous salbutamol compared with intravenous salbutamol plus ipratropium bromide). Children administered intravenous salbutamol ceased supplemental oxygen therapy earlier than those administered ipratropium alone at 12 hrs post-randomization ($p = 0.0003$). Children administered intravenous salbutamol could be discharged from the hospital 28 hrs earlier than those administered ipratropium bromide ($p = 0.013$). CONCLUSION: Children administered intravenous salbutamol for severe acute asthma showed a more rapid recovery time, which resulted in earlier discharge from the hospital than those administered inhaled ipratropium bromide. There was no additional benefit obtained by combining ipratropium bromide and intravenous salbutamol administration.

Complications of Central Venous Catheters: Internal Jugular Versus Subclavian Access—A Systematic Review—Ruesch S, Walder B, Tramer MR. Crit Care Med 2002 Feb;30(2):454-460.

OBJECTIVE: To test whether complications happen more often with the internal jugular or the subclavian central venous approach. DATA SOURCE: Systematic search (MEDLINE, Cochrane Library, EMBASE, bibliographies) up to June 30, 2000, with no language restriction. STUDY SELECTION: Reports on prospective comparisons of internal jugular vs. subclavian catheter insertion, with dichotomous data on complications. DATA EXTRACTION: No valid randomized trials were found. Seventeen prospective comparative trials with data on 2,085 jugular and 2,428 subclavian catheters were analyzed. Meta-analyses were performed with relative risk (RR) and 95% confidence interval (CI), using fixed and random effects models. DATA SYNTHESIS: In six trials (2,010 catheters), there were significantly more arterial punctures with jugular catheters compared with subclavian (3.0% vs. 0.5%, RR 4.70 [95% CI, 2.05–10.77]). In six trials (1,299 catheters), there were significantly less malpositions with the jugular access (5.3% vs. 9.3%, RR 0.66 [0.44–0.99]). In three trials (707 catheters), the incidence of bloodstream infection was 8.6% with the jugular access and 4.0% with the subclavian access (RR 2.24 [0.62–8.09]). In ten trials (3,420 catheters), the incidence of hemato- or pneumothorax was 1.3% vs. 1.5% (RR 0.76 [0.43–1.33]). In four trials (899), the incidence of vessel occlusion was 0% vs. 1.2% (RR 0.29 [0.07–1.33]). CONCLUSIONS: There are more arterial punctures but less catheter malpositions with the internal jugular compared with the subclavian access. There is no evidence of any difference in the incidence of hemato- or pneumothorax and vessel occlusion. Data on bloodstream infection are scarce. These data are from nonrandomized studies; selection bias cannot be ruled out. In terms of risk, the data most likely represent a best case scenario. For rational decision-making, randomized trials are needed.

Severe Status Asthmaticus: Management with Permissive Hypercapnia and Inhalation Anesthesia—Mutlu GM, Factor P, Schwartz DE, Sznajder JI. Crit Care Med 2002 Feb;30(2):477-480.

OBJECTIVE: To describe the difficulties that can be encountered during mechanical ventilation of severe status asthmaticus and to discuss the safety of permissive hypercapnia as a ventilatory strategy and the role and limitations of inhalation anesthesia in the treatment of refractory cases. DESIGN: Case series and review of literature. SETTING: Intensive care unit of a tertiary care hospital. PATIENTS: Two patients with severe status asthmaticus. INTERVENTIONS: Administration of inhalational anesthetics. MEASUREMENTS AND MAIN RESULTS: Both patients had respiratory failure secondary to status asthmaticus requiring mechanical ventilation and permissive hypercapnia. They also received inhalational anesthetics because of refractory bronchoconstriction. Levels of P_{aCO_2} in each case were among the highest and most prolonged elevations (>150 mm Hg for several hours) reported to date. In one case, life-threatening difficulties with ventilation were encountered related to the use of an anesthesia ventilator. Although they had complications related to the severity of their illnesses, both were treated to recovery. CONCLUSIONS: Mechanical ventilation in severe status asthmaticus can be challenging. Permissive hypercapnia is a relatively safe strategy in the ventilatory management of asthma. High levels of hypercapnia and associated severe acidosis are well tolerated in the absence of contraindications (i.e., preexisting intracranial hypertension). Inhalation anesthesia may be useful in the treatment of refractory cases of asthma but should be used carefully because it may be hazardous owing to poor flow capabilities of most anesthesia ventilators.

Effect of Long Term Treatment with Azithromycin on Disease Parameters in Cystic Fibrosis: A Randomised Trial—Wolter J, Seeney S, Bell S, Bowler S, Masel P, McCormack J. Thorax 2002 Mar;57(3):212-216.

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BACKGROUND: Relentless chronic pulmonary inflammation is the major contributor to morbidity and mortality in patients with cystic fibrosis (CF). While immunomodulating therapies such as prednisolone and ibuprofen may be beneficial, their use is limited by side effects. Macrolides have immunomodulatory properties and long term use dramatically improves prognosis in diffuse panbronchiolitis, a condition with features in common with the lung disease of CF. METHODS: To determine if azithromycin (AZM) improves clinical parameters and reduces inflammation in patients with CF, a 3 month prospective randomised double blind, placebo controlled study of AZM (250 mg/day) was undertaken in adults with CF. Monthly assessment included lung function, weight, and quality of life (QOL). Blood and sputum collection assessed systemic inflammation and changes in bacterial flora. Respiratory exacerbations were treated according to the policy of the CF Unit. RESULTS: Sixty patients were recruited (29 men) of mean (SD) age 27.9 (6.5) years and initial forced expiratory volume in 1 second (FEV_1) 56.6 (22.3)% predicted. FEV_1% and forced vital capacity (FVC)% predicted were maintained in the AZM group while in the placebo group there was a mean (SE) decline of -3.62 (1.78)% (p=0.047) and -5.73 (1.66)% (p=0.001), respectively. Fewer courses of intravenous antibiotics were used in patients on AZM (0.37 v 1.13, p=0.016). Median C reactive protein (CRP) levels declined in the AZM group from 10 to 5.4 mg/mL but remained constant in the placebo group (p<0.001). QOL improved over time in patients on AZM and remained unchanged in those on placebo (p=0.035). CONCLUSION: AZM in adults with CF significantly improved QOL, reduced CRP levels and the number of respiratory exacerbations, and reduced the rate of decline in lung function. Long term AZM may have a significant impact on morbidity and mortality in patients with CF. Further studies are required to define frequency of dosing and duration of benefit.

Attack Context: An Important Mediator of the Relationship Between Psychological Status and Asthma Outcomes—Greaves CJ, Eiser C, Seamark D, Halpin DM. Thorax 2002 Mar;57(3):217-221.

BACKGROUND: The importance of psychosocial variables in asthma is increasingly recognised, although attempts to relate these to asthma outcomes often produce only weak relationships. This study aimed to identify whether such relationships might be obscured by the effects of recent asthma experience on psychological status. METHODS: An adult community sample of 37 patients who had suffered a recent attack of asthma and 37 with stable asthma were given measures of panic fear and control confidence. The relationship with subsequent emergency service use was examined using two way ANOVA and correlational analyses. Covariate influences (psychiatric morbidity, age, sex, treatment level, asthma duration, social status) were considered. RESULTS: Control confidence predicted emergency service use in different ways for recent attack and stable asthma patients. This interaction was highly significant ($F(1,69) = 10.32$, $p<0.005$) with high confidence relating to an increased risk of an attack in the recent attack group and low confidence relating to increased risk for the stable asthma group. There was also an interaction between panic fear and attack context ($F(1,69) = 11.05$, $p<0.005$) with low panic fear resulting in more attacks for recent attack cases. CONCLUSIONS: Attack context (having a recent attack) is an important mediator of psychological status. Strong cognitive/affective responses to attacks may motivate improved self-care and this represents a window of opportunity for self-care interventions. Weak cognitive/affective responses to attacks may reflect denial and require different intervention approaches. For those with recently stable asthma the relationships are qualitatively and quantitatively different, and the implications for intervention are also discussed.

Laser Acupuncture in Children and Adolescents with Exercise Induced Asthma—Gruber W, Eber E, Malle-Scheid D, Pfleger A, Weinhandl E, Dorfer L, Zach MS. Thorax 2002 Mar;57(3):222-225.

Background: Laser acupuncture, a painless technique, is a widely used alternative treatment method for childhood asthma, although its efficacy has not been proved in controlled clinical studies. Methods: A double blind, placebo controlled, crossover study was performed to investigate the possible protective effect of a single laser acupuncture treatment on cold dry air hyperventilation induced bronchoconstriction in 44 children and adolescents of mean age 11.9 years (range 7.5–16.7) with exercise induced asthma. Laser acupuncture was performed on real and placebo points in random order on two consecutive days. Lung function was measured before laser acupuncture, immediately after laser acupuncture (just before cold dry air challenge (CACh)), and 3 and 15 minutes after CACh. CACh consisted of a 4 minute isocapnic hyperventilation of −10C absolute dry air. Results: Comparison of real acupuncture with placebo acupuncture showed no significant differences in the mean maximum CACh induced decrease in forced expiratory volume in 1 second (27.2 (18.2)% v 23.8 (16.2)%) and maximal expiratory flow at 25% remaining vital capacity (51.6 (20.8)% v 44.4 (22.3)%). Conclusions: A single laser acupuncture treatment offers no protection against exercise induced bronchoconstriction in paediatric and adolescent patients.

Influence of Cigarette Smoking on Inhaled Corticosteroid Treatment in Mild Asthma—Chalmers GW, Macleod KJ, Little SA, Thomson LJ, McSharry CP, Thomson NC. Thorax 2002 Mar;57(3):226-230.

Background: Although inhaled corticosteroids have an established role in the treatment of asthma, studies have tended to concentrate on non-smokers and little is known about the possible effect of cigarette smoking on the efficacy of treatment with inhaled steroids in asthma. A study was undertaken to investigate the effect of active cigarette smoking on responses to treatment with inhaled corticosteroids in patients with mild asthma. Methods: The effect of treatment with inhaled fluticasone propionate (1000 microg daily) or placebo for 3 weeks was studied in a double blind, prospective, randomised, placebo controlled study of 38 steroid naive adult asthmatic patients (21 non-smokers). Efficacy was assessed using morning and evening peak expiratory flow (PEF) readings, spirometric parameters, bronchial hyperreactivity, and sputum eosinophil counts. Comparison was made between responses to treatment in non-smoking and smoking asthmatic patients. Results: There was a significantly greater increase in mean morning PEF in non-smokers than in smokers following inhaled fluticasone (27 L/min v −5 L/min). Non-smokers had a statistically significant increase in mean morning PEF (27 L/min), mean forced expiratory volume in 1 second (0.17 L), and geometric mean PC_{20} (2.6 doubling doses), and a significant decrease in the proportion of sputum eosinophils (−1.75%) after fluticasone compared with placebo. No significant changes were observed in the smoking asthmatic patients for any of these parameters. Conclusions: Active cigarette smoking impairs the efficacy of short term inhaled corticosteroid treatment in mild asthma. This finding has important implications for the management of patients with mild asthma who smoke.

Inspiratory Muscle Maximum Relaxation Rate Measured from Submaximal Sniff Nasal Pressure in Patients with Severe COPD—Kyroussis D, Johnson LC, Hamnegard CH, Polkey MI, Moxham J. Thorax 2002 Mar;57(3):254-257.

BACKGROUND: Slowing of the inspiratory muscle maximum relaxation rate (MRR) is a useful index of severe inspiratory muscle loading and potential fatigue and has been measured from the oesophageal pressure during sniffs in patients with chronic obstructive pulmonary disease (COPD). The purpose of this study was to investigate whether it is possible to measure MRR and detect slowing using sniff nasal pressure in patients with COPD and to investigate the relationship between sniff oesophageal and sniff nasal MRR. METHODS: Eight patients with severe COPD (mean FEV_1 0.7 l; 26% predicted) were studied. Each subject performed submaximal sniff manoeuvres before and after walking to

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a state of severe dyspnoea on a treadmill. Oesophageal and gastric pressures were measured using balloon tipped catheters and nasal pressure was measured using an individually modelled nasal cast. MRR (% pressure fall/10 ms) was determined for each sniff and any change following exercise was reported as percentage of baseline to allow comparison of sniff nasal and oesophageal MRR. RESULTS: At rest the mean (SE) sniff P_{oes} MRR was 7.1 (0.3) and the mean P_{nasal} MRR was 8.6 (0.1). At 1 minute following exercise there was a mean decrease in sniff P_{oes} MRR of 33.7% (range 20.7-53.4%) and a mean decrease in sniff P_{nasal} MRR of 28.2% (range 8.1-52.8%). The degree of slowing and time course of recovery was similar, with both returning to baseline values within 5-10 minutes. A separate analysis of the sniff pressures using only the nasal pressure traces demonstrated a similar pattern of slowing and recovery. CONCLUSIONS: It is possible to detect slowing of the inspiratory muscles non-invasively using sniff nasal pressures in patients with COPD. This could be a useful technique with which to measure severe and potentially fatiguing inspiratory muscle loading, both in clinical settings and during exercise studies.

Effect of Assist Negative Pressure Ventilation by Microprocessor Based Iron Lung on Breathing Effort—Gorini M, Villella G, Ginanni R, Augustynen A, Tozzi D, Corrado A. Thorax 2002 Mar;57(3):258-262.

Background: The lack of patient triggering capability during negative pressure ventilation (NPV) may contribute to poor patient synchrony and induction of upper airway collapse. This study was undertaken to evaluate the performance of a microprocessor based iron lung capable of thermistor triggering. Methods: The effects of NPV with thermistor triggering were studied in four normal subjects and six patients with an acute exacerbation of chronic obstructive pulmonary disease (COPD) by measuring: (1) the time delay (TDtr) between the onset of inspiratory airflow and the start of assisted breathing; (2) the pressure-time product of the diaphragm (PTPdi); and (3) non-triggering inspiratory efforts (NonTrEf). In patients the effects of negative extrathoracic end expiratory pressure (NEEP) added to NPV were also evaluated. Results: With increasing trigger sensitivity the mean (SE) TDtr ranged from 0.29 (0.02) s to 0.21 (0.01) s (mean difference 0.08 s, 95% CI 0.05 to 0.12) in normal subjects and from 0.30 (0.02) s to 0.21 (0.01) s (mean difference 0.09 s, 95% CI 0.06 to 0.12) in patients with COPD; NonTrEf ranged from 8.2 (1.8)% to 1.2 (0.1)% of the total breaths in normal subjects and from 11.8 (2.2)% to 2.5 (0.4)% in patients with COPD. Compared with spontaneous breathing, PTPdi decreased significantly with NPV both in normal subjects and in patients with COPD. NEEP added to NPV resulted in a significant decrease in dynamic intrinsic PEEP, diaphragm effort exerted in the pre-trigger phase, and NonTrEf. Conclusions: Microprocessor based iron lung capable of thermistor triggering was able to perform assist NPV with acceptable TDtr, significant unloading of the diaphragm, and a low rate of NonTrEf. NEEP added to NPV improved the synchrony between the patient and the ventilator.

The Pulmonary Physician in Critical Care # 3: Critical Care Management of Community Acquired Pneumonia—Baudouin SV. Thorax 2002 Mar;57(3):267-271.

Severe community acquired pneumonia carries a high mortality. Early recognition of the severity of the illness, rapid and appropriate resuscitation, targeted antibiotic treatment, and the critical care support of multiple failing organ systems are all important in this group of patients. Only by improving all these aspects of care is it likely that survival will increase.

Proportional Assist Ventilation (PAV): A Significant Advance or a Futile Struggle Between Logic and Practice?—Ambrosino N, Rossi A. Thorax 2002 Mar;57(3):272-276.

Proportional assist ventilation is a promising addition to other more conventional modes of mechanical ventilation with the theoretical advantage of improving patient-ventilator interaction. It may also be of use as a diagnostic tool in the control of breathing in mechanically ventilated patients.

Bronchodilating Effect of Oxitropium Bromide in Heart Disease Patients with Exacerbations of COPD: Double-Blind, Randomized, Controlled Study—Centanni S, Santus P, Casanova F, Carlucci P, Boveri B, Castagna F, et al. Respir Med 2002 Mar;96(3):137-141.

Anti-cholinergic agents are considered the bronchodilator therapy of first-choice in the treatment of patients with stable chronic obstructive pulmonary disease (COPD) associated with heart disease since they may be as effective or more effective than inhaled β_2-agonists and, moreover, they do not interact with cardiac β-adrenoceptors. The aim of our study was to evaluate the bronchodilator activity of oxitropium bromide in outpatients suffering from exacerbations of COPD associated with heart diseases (ischaemic heart disease and/or arrhythmias). We recruited 50 consecutive outpatients (33 males and 17 females, mean age 68.6 years, 15 current smokers and 35 ex-smokers). Each patient performed body plethismography in basal condition and 30 min after inhalation of 200 microg metered dose inhaler (MDI) oxitropium bromide administered by a device (Fluspacer). FEV_1, FVC, $MMEF_{25-75}$, sR_{aw} and tR_{aw} were evaluated. Thirty minutes after 200 microg oxitropium bromide administration, we observed a significant improvement in FEV_1 11.6% ± 1 (mean ± SEM) ($p<0.01$); FVC, $MMEF_{25-75}$ sR_{aw} variation was respectively: 9.2% ± 0.6, 31.4 ± 2.9, -19.9 ± 1.1. Placebo did not significantly change pulmonary function. Our data suggest that oxitropium bromide bronchodilator activity is effective in exacerbations of COPD.

Asthma Control and Differences in Management Practices Across Seven European Countries—Vermeire PA, Rabe KF, Soriano JB, Maier WC. Respir Med 2002 Mar;96(3):142-149.

Failure to follow asthma management guidelines may result in poor asthma control for many patients. The Asthma Insights and Reality in Europe (AIRE) survey, a multi-national survey assessing the level of asthma control from the patients perspective in seven Western European countries, previously demonstrated that the Global Initiative for Asthma (GINA) guideline goals were not achieved in Western Europe and that both adults and children with asthma were poorly controlled. Using additional data on asthma management practices from each of the seven countries in the AIRE survey, we compared variations in asthma morbidity and asthma management practices across countries to provide insight into the reasons for poor asthma control. Asthma management practices and asthma control among adults and children with current asthma were suboptimal in each of seven countries surveyed. Among patients with symptoms of severe persistent asthma, over 40% reported their asthma was well or completely controlled. School absence due to asthma was reported by up to 52.7% of children and up to 27.6% of adult reported work absence due to asthma. Lung function testing in the past year was uncommon: ranging from 13.5% of children in the U.K. to 68.8% of adults in Germany. Written asthma management plans were used by less than 50% of adults and less than 61% of children in all seven countries. Most adults (49.5-73.0%) and a large proportion of children (38.4-70.6%) had follow-up visits for their asthma only when problems developed. The ratio of recent inhaled corticosteroid use to recent short-acting ß-agonist use was inappropriate (<1) among patients with symptoms of severe asthma in all countries. This disparity was greatest among adults in Italy and France, where recent inhaled corticosteroid use was reported by less than one in nine patients reporting recent use of short-acting bronchodilators (IS:SAB <0.11). Management practices differ between countries and additional public health interventions and resources may be necessary to reduce patient suffering. Further efforts to fully implement asthma management guidelines are required to improve asthma control in Europe.

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The Use of the Air Leak Test and Corticosteroids in Intubated Children: A Survey of Pediatric Critical Care Fellowship Directors

Jason A Foland MD, Dennis M Super MD MPH, Nagib S Dahdah MD, and Maroun J Mhanna MD

INTRODUCTION: Uncertainties exist regarding the value of the air leak test or use of steroids for preventing post-extubation stridor and extubation failure in children. OBJECTIVE: Determine the practice preferences of pediatric critical care physicians regarding the air leak test and administration of glucocorticosteroids to prevent airway edema. METHODS: A 14-question survey regarding the value of the air leak test, use of glucocorticosteroids, and management of airway edema in intubated children was sent to all North American pediatric critical care fellowship directors affiliated with medical school teaching hospitals. RESULTS: The response rate was 85% (58/68). Seventy-six percent (44/58) routinely check for air leak prior to extubation. The physicians who check for air leak were more likely to delay extubation in order to administer glucocorticosteroids (60% [26/43] vs 15% [2/13], p = 0.01). An air leak of ≥ 30 cm H_2O was more likely (than ≥ 20 cm H_2O) to result in delaying extubation (95% [35/37] vs 51% [19/37], p <0.001). Of the respondents who use steroids for airway edema prophylaxis, 73% (24/33) give steroids based on the air leak test. CONCLUSIONS: The majority of surveyed pediatric critical care fellowship program directors rely on the air leak test and use corticosteroids to prevent post-extubation stridor and extubation failure. At an air leak of ≥ 30 cm H_2O most of the surveyed physicians would delay extubation and initiate glucocorticosteroids. *Key words: pediatrics, intensive care unit, steroids, health care survey, stridor, air leak test, extubation.* [Respir Care 2002;47(6):662–666]

Introduction

Tracheal intubation can result in damage to the larynx, manifested by inflammation and swelling, leading to complications such as stridor and upper airway obstruction.[1–4] In neonates and infants the presence of post-extubation stridor and the number of endotracheal tube (ETT) insertions directly correlate with the risk of developing subglottic stenosis.[5–7]

The air leak test measures the air pressure required to produce an audible (to a stethoscope placed over the larynx) rush of air around the ETT.[8–10] The air leak test has been used to predict successful extubation among children with viral croup and after tracheal surgery—conditions that can produce upper airway edema.[11–13] It has become a common practice in many centers to routinely test for air leak prior to extubation in all intubated children, regardless of the underlying pathology. A study by Kemper et al of pediatric trauma patients found that the absence of an

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air leak at 30 cm H_2O pressure might predict extubation failure.[14]

The utility of steroids in reducing post-extubation laryngeal edema is uncertain. A single dose of hydrocortisone prior to extubation showed no beneficial effect in adults, whereas in pre-term infants dexamethasone prevented post-extubation airway obstruction.[15,16] Studies of the use of dexamethasone to prevent airway obstruction and post-extubation stridor in children have given mixed results.[17–19] Children in certain age groups are more susceptible to post-intubation laryngeal edema, yet attempts to stratify children by age showed no advantage from dexamethasone in preventing post-extubation stridor.[4,7,17] Uncertainty also exists regarding steroid dosing regimen prior to extubation.[15–18]

The use of cuffed ETTs in children < 7 years of age has not been recommended by the Pediatric Advanced Life Support Committee of the American Heart Association, but it has been a common practice to use cuffed ETTs in young children.[20,21] ETT size may affect airway edema. Whether down-sizing the ETT in the absence of an air leak decreases the development of airway edema or stridor is unknown.

Our objective was to determine areas of consensus and controversy among pediatric intensivists in the management of airway edema. A questionnaire was distributed to all North American pediatric critical care fellowship directors affiliated with medical school teaching hospitals. The survey was conducted to assess practice patterns in areas of controversy, and the information generated from our survey will provide data to frame future research questions and improve our current practice.

Methods

Our institutional review board approved the protocol for this survey. All pediatric critical care fellowship directors registered with the American Academy of Pediatrics were surveyed. The directors were initially recruited by an electronic-mail survey. Electronic mail and postal addresses were obtained from the Pediatric Critical Care Web site (http://PedsCCM.org). Nonrespondents received 2 follow-up e-mails, one week apart. Physicians who did not respond to the e-mails received an additional mailing via the postal service, and if they did not respond to that mailing they were considered nonresponders. All returned surveys were included in the results, even if the questionnaire was not complete. Respondents' demographic data was obtained from the Web sites of the American Board of Pediatrics and the American Academy of Pediatrics.

The survey addressed individual practices of pediatric critical care fellowship directors regarding the value of the air leak test, use of glucocorticosteroids, and management of airway edema in intubated children. Figure 1 shows the survey questionnaire.

Statistical Analysis

For dichotomous data, the Fisher's exact test was used for unpaired data, and McNemar's test was used for paired data. Categorical data are reported as percentages, with the corresponding 95% confidence interval.[22] Statistical significance was defined a priori as $p < 0.05$, 2-tail.

Results

The response rate was 85% (58/68). Seventy-two percent (42/58) of the respondents completed the questionnaire. The remainder answered at least 11 of the 14 questions.

In response to the questions about the air leak test (questions 1 and 2), 76% (44/58, 95% confidence interval 62.6, 85.7) of the respondents check for air leak prior to extubation. The demographics were similar among physicians who do and do not check for air leak, regarding the number of years since medical school graduation, pediatric critical care board certification, gender, primary specialty, and board certification. Among the physicians who check for air leak, 37 reported their definition of an abnormal test. A leak pressure < 20 cm H_2O was considered safe for extubation by all of the respondents, whereas almost half considered a value < 30 cm H_2O safe for extubation (100% [37/37] versus 49% [18/37], $p < 0.001$). Only 5% (2/37) of respondents considered extubation safe if the air leak value was < 40 cm H_2O. An air leak value > 30 cm H_2O (versus ≥ 20 cm H_2O) was more likely to result in delaying extubation (95% [35/37] versus 51% [19/37], $p < 0.001$).

In response to the questions about the use of steroids (questions 3–12), 19% (11/57) of all respondents routinely use steroids for airway edema prophylaxis. Table 1 shows the response to the question regarding extubation delay after administration of steroids for an abnormal air leak test. Table 2 shows the response to the question about the use of steroids based on air leak values.

In response to the question regarding the use of steroids for airway edema prophylaxis in children of different age groups, 81% (29/36) do not consider age in the decision to use steroids. Only 14% (5/36) consider using steroids in children < 1 year of age, regardless of the air leak level.

In response to the question about pre-extubation steroid regimen, 13% (6/48) use a single dose, 31% (15/48) use 2 doses, 27% (13/48) use 3 doses, and 29% (14/48) use 4 doses. All of the respondents (50/50) who use corticosteroids would use dexamethasone for airway edema prophylaxis. Overall, 46% (26/56) of all respondents check for air leak, administer steroids, and delay extubation, whereas

1. Do you routinely check for an air leak around the endotracheal tube before extubation? (yes or no)

2. If you check for an air leak before extubation, what level would you consider safe for extubation?
   a) < 20 cm H_2O
   b) < 30 cm H_2O
   c) < 40 cm H_2O
   d) Do not check air leak

3. Do you routinely use steroids for airway edema prophylaxis before extubation? (yes or no)

4. How long would you delay extubation if steroids were started for an abnormal air leak test?
   a) No delay
   b) < 6 hours
   c) 6–12 hours
   d) 12–24 hours
   e) > 24 hours
   f) Do not use steroids

5. If you use steroids for airway edema prophylaxis before extubation, at what air leak level would you start them?
   a) Give regardless of air leak level
   b) ≥ 20 cm H_2O
   c) ≥ 25 cm H_2O
   d) ≥ 30 cm H_2O
   e) ≥ 35 cm H_2O
   f) ≥ 40 cm H_2O
   g) Do not use steroids

6. If you use steroids for airway edema prophylaxis before extubation, do you start them regardless of air leak levels in certain ages? (more than one answer possible)
   1. < 1 year
   2. 1–5 years
   3. 5–10 years
   4. > 10 years
   5. I use steroids, but age is not a factor
   6. Do not use steroids

7. How many doses of steroids would you give for airway edema prophylaxis before extubation?
   a) 1 dose
   b) 2 doses
   c) 3 doses
   d) 4 doses
   e) Do not use steroids

8. If you use steroids for airway edema prophylaxis before extubation, which one do you use?
   a) Dexamethasone
   b) Methylprednisone
   c) Hydrocortisone
   d) Other (please name)
   e) Do not use steroids

9. In your opinion, do steroids reduce the risk of post-extubation stridor? (yes or no)

10. In your opinion, do steroids reduce the risk of extubation failure (re-intubation) pertaining to airway edema? (yes or no)

11. Do you routinely use steroids for airway edema prophylaxis before extubation if a patient was intubated for viral croup? (yes or no)

12. Do you routinely use steroids for airway edema prophylaxis before extubation if a patient has had upper airway and/or tracheal reconstructive surgery? (yes or no)

13. If no air leak is noted and the patient is not ready for extubation, do you electively change the endotracheal tube to a smaller size? (yes or no)

14. Do you use cuffed endotracheal tubes in children less than 7 years old? (yes or no)

Fig. 1. Survey questionnaire.

only 20% (11/56) do not check for air leak, administer steroids, or delay extubation (Figure 2).

Eighty percent (44/55) and 78% (43/55) of all the respondents consider steroids effective in preventing post-extubation stridor and extubation failure, respectively. Seventy-two percent (42/58) use corticosteroids prophylactically for children intubated due to viral croup, and 74% (43/58) use corticosteroids for patients intubated after airway surgery.

Forty-two percent (24/57) of all the respondents would down-size the ETT in the absence of an air leak in intubated patients not ready for extubation, and 74% (43/58) use cuffed ETTs in children < 7 years of age.

Discussion

The majority of the responding pediatric critical care fellowship directors check for air leak and use steroids to prevent post-extubation stridor and extubation failure. At an air leak ≥ 30 cm H_2O, most respondents would delay extubation and use steroids. Almost half of our respondents also would down-size the ETT in the absence of an air leak in patients not ready for extubation, and 74% use cuffed ETTs in children < 7 years old.

Post-extubation laryngeal edema can cause upper airway obstruction.[1–3] In adults the air leak test is equivocal as a predictor of post-extubation stridor or airway obstruction.[8,13,23] In children the air leak test has been predictive of post-extubation outcome in patients with viral croup or after tracheal surgery.[11,12] In children intubated for acute viral croup an air leak pressure < 40 cm H_2O successfully predicted extubation outcome.[11] An air leak level ≤ 20 cm H_2O predicted successful extubation following tracheal surgery (sensitivity 100%), whereas a level > 30 cm H_2O predicted extubation failure.[12] In their study of pediatric trauma patients, Kemper et al advocated the 30 cm H_2O cutoff level. In that study, air leak levels > 30 cm H_2O

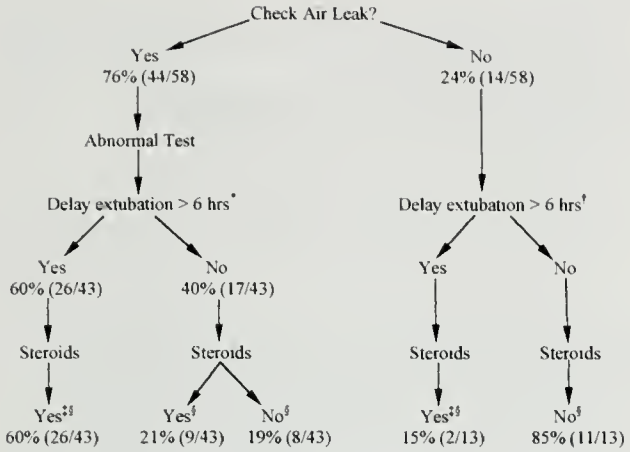

Fig. 2. Steroid use based on air leak test results. *One physician in the group that checks for air leak had an incomplete response to this question. † One physician in the group that does not check for air leak had an incomplete response to this question. ‡ Sixty percent (26/43) of the physicians who use steroids and check for air leak delay extubation, versus 15% (2/13) of the physicians who use steroids and do not check for air leak ($p = 0.01$). § Seventy-six percent (28/37) of the physicians who use steroids delay extubation, whereas none (0/19) of the physicians who do not use steroids delay extubation ($p < 0.001$).

were associated with airway edema serious enough to create post-extubation stridor (sensitivity 100%).[14] Respondents to our survey use air leak pressures of 20 cm H_2O and 30 cm H_2O as criteria for extubation, regardless of the underlying pathology. However, in our questionnaire we used only 20 cm H_2O and 30 cm H_2O as cutoff points, so we may have missed the opinions of those who use other values such as 25 or 35 cm H_2O.

Dexamethasone has been shown by several investigators to be beneficial in reducing stridor from infectious croup.[2-4,25] It is controversial whether dexamethasone is effective in preventing post-extubation complications due to airway edema.[17-19] However, the majority of our respondents reported that corticosteroids are effective in reducing the incidence of post-extubation stridor and extubation failure secondary to airway edema.

Previous studies indicated that 2 doses of dexamethasone (taken 6 hours apart) decreases the incidence of post-extubation airway obstruction in pre-term infants.[16] In children, however, studies using 6 doses of dexamethasone (taken 1 dose every 6 hours) to prevent airway obstruction and post-extubation stridor have yielded conflicting results.[17] Most of our respondents give 2 or more doses of steroids prior to extubation, regardless of the patient's age. In our questionnaire we did not ask about the dose (in mg/kg) of steroids used to prevent post-extubation complications. It would be of interest to know if all participants use the same dose of dexamethasone.

Table 1. Percentage of Physicians Who Would Delay Extubation in Order to Use Steroids

Delay (h)	% Physicians
No Delay	17 (8/46)
< 6	11 (5/46)
6–12	15 (7/46)
12–24	33 (15/46)
> 24	24 (11/46)

Table 2. Percentage of Physicians Who Would Use Steroids Based on Air Leak Values*

Air Leak Value (cm H_2O)	% Reply	Cumulative %
≥ 20	25 (6/24)	25 (6/24)
≥ 25	17 (4/24)	42 (10/24)
≥ 30	50 (12/24)	92 (22/24)
≥ 35	4 (1/24)	96 (23/24)
≥ 40	4 (1/24)	100 (24/24)

*Nine physicians use steroids regardless of the air leak value.

The use of a large-diameter-for-age ETT may lead to an abnormal air leak test and airway edema.[26] Our respondents had different answers regarding the management of patients with large-diameter-for-age ETTs and absence of an air leak. Less than half would consider down-sizing the ETT in hopes of reducing airway edema.

The use of cuffed ETTs in children < 7 years of age is controversial[20] because it is believed that the normal narrowing of the cricoid cartilage provides a natural cuff, and a cuffed ETT may cause subglottic edema and stenosis.[21] The majority of our respondents use cuffed ETTs in young children, which is consistent with the recommendations of Deakers et al, who have shown the safety of cuffed ETTs in young children.[20]

A potential limitation to our study is that we only surveyed pediatric critical care fellowship program directors. That cohort was chosen because it may represent the opinions of academic physicians in various teaching hospitals and may reflect the common practice and teaching that is passed on to pediatric critical care fellows in training. It is quite possible that the practice of other members of the academic group and physicians in private practice is different from the fellowship directors' opinions. Hence, the views of only 58 pediatric critical care fellowship directors may not fully represent the practice of the pediatric critical care community. However, our study was strengthened by an 85% response rate. By surveying all the centers with fellowship programs in both the United States and Canada, and by surveying only fellowship directors, we reduced the risk of having the clinical practice of one center over-represented in this survey.

Additionally, this study was a survey and it is subject to a bias imposed by the questionnaire choices. The survey did not include open-ended questions. The purpose of our study was not to determine the utility of the air leak test or use of steroids but to determine the common practices of pediatric critical care physicians. The information generated from this survey highlights the need for future studies to evaluate the utility of the air leak test and corticosteroids to prevent post-extubation stridor and failure.

Conclusions

Our study shows that the majority of pediatric critical care fellowship directors check for air leak and use steroids prior to extubation in order to prevent post-extubation stridor and extubation failure. Most of the pediatric critical care fellowship directors delay extubation if the air leak is $\geq$ 30 cm H_2O, and the majority also delay extubation in order to administer 2 or more doses of steroids. Additional studies are needed to ascertain which patients are at risk for airway edema and to evaluate treatment strategies that may reduce post-extubation stridor and the need for reintubation.

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Imposed Work of Breathing During Ventilator Failure

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INTRODUCTION: Ventilators possess an anti-asphyxia valve that allows spontaneous breathing of ambient air during ventilator failure. This study examined the imposed work of breathing and pressure-time product of 8 critical care and 9 portable ventilators, using a laboratory simulation of spontaneous breathing during ventilator failure. METHODS: A test lung was modified to simulate spontaneous breathing with a tidal volume of 0.5 L and peak inspiratory flow of 60 L/min. A pneumotachograph and pressure tap were placed at the proximal airway between the breathing circuit and endotracheal tube. Flow was derived from the pressure drop across the pneumotachograph. Signals were amplified, integrated, and saved to a spreadsheet program, and imposed work of breathing and pressure-time product were calculated. Also measured were the inspiratory pressure required to open the anti-asphyxia valve (cracking pressure), time to cracking pressure, maximum negative inspiratory pressure, and time to maximum negative inspiratory pressure. RESULTS: For the critical care ventilators the mean ± SD imposed work of breathing ranged from 213.07 ± 3.53 to 890.63 ± 0.88 mJ/L and the pressure-time product ranged from 2.67 ± 0.01 to 13.37 ± 0.01 cm H_2O · s/L. For the portable ventilators the mean ± SD imposed work of breathing ranged from 361.37 ± 1.22 to 969.60 ± 22.70 mJ/L and the pressure-time product ranged from 4.52 ± 0.01 to 16.70 ± 0.37 cm H_2O · s/L. CONCLUSIONS: Spontaneous breathing during ventilator failure may impose work approximating the physiologic work of breathing. This imposed work may prevent effective breathing through the anti-asphyxia valve during mechanical ventilator failure due to electrical failure. These results reinforce the need to properly monitor mechanically ventilated patients and to have in place sufficient back-up power supplies and a method of manual ventilation. *Key words: imposed work of breathing, ventilator failure, anti-asphyxia valve, pressure-time product, ventilator, test lung, cracking pressure, electrical failure.* [Respir Care 2002;47(6):667–674]

Introduction

Minimizing the imposed work of breathing (WOB_I) is a principal goal of mechanical ventilation as well as of mechanical ventilator design. Numerous investigators have evaluated the WOB_I characteristics of ventilators during simulated spontaneous breathing.[1–9] During our previous evaluation of ventilator operation we noted significant differences in WOB_I characteristics of ventilators following power failure.[7–11] Ventilators designed for transport and home care and, more recently, critical care ventilators, are equipped with battery back-up to continue operation in the event of power failure. Additionally, American Society of Testing and Materials standards (ASTM standards F1100 and F1246)[12,13] require ventilators to incorporate a mechanical safety valve, often referred to as an anti-asphyxia

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valve, that allows spontaneous breathing from ambient air in the event of power (electric or compressed gas) failure. Though failure of mechanical ventilators is rare, at least one case of ventilator malfunction during power failure has been described.[14–17]

Given the variety of environments in which ventilators are used and the possibility of power loss, we conducted a bench study to evaluate WOB imposed by anti-asphyxia valves in both portable and critical care ventilators.

Alarms should indicate ventilator failure, and ventilator failure should be immediately detected in a critical care setting. However, it is not inconceivable that a substantial period of time could elapse between ventilator failure and detection in a subacute or home setting. Thus, patients in those settings may be required to breathe via the anti-asphyxia valve for several minutes, and it is important that the WOB_I be easily tolerated by the ventilator-dependent patient.

Methods

Experimental Setup

Spontaneous breathing was simulated using a model similar to that described by Op't Holt et al.[18] A 2-chambered test lung (TTL, Michigan Instruments, Grand Rapids, Michigan) was modified to allow simulation of spontaneous breathing. One chamber (the driving chamber) was attached to a ventilator (Evita 4, Dräger Medical, Telford, Pennsylvania). The ventilator to be tested was attached to the other chamber (the test chamber). The breathing gas was not humidified. A metal lift bar was attached to the driving chamber so that, when ventilated, the driving chamber lifted the test chamber, creating sub-atmospheric pressure in the test chamber.

Compliance of both chambers was set at 0.05 L/cm H_2O. A 5 cm H_2O/L/s parabolic resister (TTL, Michigan Instruments, Grand Rapids, Michigan) was placed between the test chamber and the pneumotachograph (Fleisch pneumotachograph, Hans Rudolph, Kansas City, Missouri). The driving ventilator simulated spontaneous breathing at 12 breaths/min, using a constant flow pattern, tidal volume (V_T) of 0.5 L, and peak inspiratory flow of 60 L/min. An uncut, 8.0 mm internal diameter endotracheal tube (Nellcor & Puritan-Bennett/Tyco Healthcare, St Louis, Missouri) was interposed between the ventilator and the test lung.

Measurements

The pneumotachograph and pressure tap were placed at the proximal airway, between the ventilator circuit and endotracheal tube. Flow was derived from the pressure drop across the pneumotachograph. Signals were amplified and integrated with a laptop computer and commercially available software (LabView, National Instruments, Austin, Texas) and saved to a spreadsheet (Excel, Microsoft, Redmond, Washington). Data were acquired at a frequency of 100 Hz. A calibrated super-syringe and U-tube manometer were used to calibrate the pneumotachograph and pressure transducer, respectively, across the range of measurement.

WOB_I (in mJ/L) was calculated as the area subtended by the pressure-volume curve.[19] Imposed pressure-time product (PTP_I in cm $H_2O \cdot$ s/L), a gauge of metabolic work performed by the respiratory muscles, was calculated as the average airway pressure multiplied by the integrated inspiratory time.[20] Cracking pressure (which is the term used in ASTM standard F1246–91) is the inspiratory pressure required to open the anti-asphyxia valve, and for this study cracking pressure was defined as the airway pressure (in cm H_2O) when an inspiratory volume of 10 mL was measured by the pneumotachograph. The value 10 mL was selected because it is easily measurable and, based on preliminary work, the anti-asphyxia valve opens when 10 mL is measured by the pneumotachograph. Time to cracking pressure was defined as the time (in ms) elapsed between the moment when airway pressure decreased from baseline to the moment when cracking pressure was attained. The maximum inspiratory pressure (P_{Imax}) was defined as the most negative pressure during inspiration. Time to P_{Imax} was defined as the time (in ms) elapsed between the moment when airway pressure decreased from baseline and the moment of most negative pressure during inspiration.

The ventilators were selected based on their availability and use in critical care, transport, subacute, and home care settings. The ventilators tested were:

- Evita 4 and Oxylog 2000 (Dräger Medical, Telford, Pennsylvania)
- 8400st, Avian, and Tbird (VIASYS Healthcare/Bird Products, Palm Springs, California)
- 7200ae, Achieva, and LP 10 (Nellcor & Puritan-Bennett/Tyco Healthcare, St Louis, Missouri)
- Amadeus, Galileo, and Veolar (Hamilton Medical, Reno, Nevada)
- Univent 750 and Univent 754 (Impact Instrumentation, West Caldwell, New Jersey)
- HT 50 (Newport Medical Instruments, Newport Beach, California)
- LTV 1000 (Pulmonetic Systems, Colton, California)
- Siemens 300 (Siemens Medical Systems, Danvers, Massachusetts)

Each ventilator was calibrated according to the manufacturer's specifications prior to each test. For the critical care ventilators the same 72-inch corrugated parallel-limb breathing circuit (Hudson RCI, Temecula, California) was used with each ventilator. The supplied or recommended breathing circuit was used with the portable ventilators.

Table 1. Cracking Pressure, Time to Cracking Pressure, Maximum Negative Inspiratory Pressure, and Time to Maximum Negative Inspiratory Pressure During Simulated Spontaneous Breathing* with Failure of 8 Critical Care Ventilators

Ventilator or Endotracheal Tube	Cracking Pressure (cm H_2O)	Time to Cracking Pressure (ms)	Maximum Negative Inspiratory Pressure (cm H_2O)	Time to Maximum Negative Inspiratory Pressure (ms)
8.0 mm ID endotracheal tube only	−0.04 ± 0	40 ± 0	−0.06 ± 0	270 ± 0
2-limb circuit and 8.0 mm ID endotracheal tube only	−0.53 ± 0	80 ± 0	−0.79 ± 0	320 ± 0
7200ae	−1.36 ± 0.02	90 ± 4.47	−2.82 ± 0.02	435 ± 38.34
8400st	−2.19 ± 0.03	100 ± 4.47	−5.83 ± 0.03	466 ± 21.91
Amadeus	−5.90 ± 0.03	130 ± 5.47	−7.46 ± 0.04	444 ± 21.91
Evita 4	−1.70 ± 0.02	100 ± 5.47	−4.70 ± 0.03	412 ± 43.82
Galileo	−2.43 ± 0.03	100 ± 4.47	−7.22 ± 0.02	458 ± 7.55
Siemens 300	−1.41 ± 0.02	100 ± 4.47	−3.37 ± 0.02	466 ± 21.91
Tbird	−2.84 ± 0.02	100 ± 4.47	−13.77 ± 0.02	406 ± 5.48
Veolar	−2.53 ± 0.02	100 ± 4.47	−5.35 ± 0.02	410 ± 27.39

*Tidal volume 0.5 L, peak inspiratory flow 1 L/s, respiratory rate 12 breaths/min.
ID = internal diameter.
Values are mean ± SD.

Each ventilator was attached to the lung simulator with the power switch in the *off* position and was not connected to a compressed gas source.

Data were collected for 5 breaths at each study condition and the means and standard deviations were calculated. Further statistical analysis was not conducted because there was little variance in the measured values.

Results

Tables 1 and 2 show the results for cracking pressure, P_{Imax}, and times to cracking pressure and P_{Imax}. Tables 3 and 4 show the results for WOB_I and PTP_I. Figures 1 and 2 show the pressure-volume curves for the critical care and portable ventilators, respectively.

For the critical care ventilators the least negative cracking pressure (–1.36 cm H_2O) was with the 7200ae. The most negative cracking pressure (–5.90 cm H_2O) was with the Amadeus. The times to cracking pressure were similar among the critical care ventilators, ranging from 90 ms (7200ae) to 130 ms (Amadeus). The least negative P_{Imax} (–2.82 cm H_2O) was with the 7200ae. The most negative P_{Imax} (–13.77 cm H_2O) was with the Tbird.

Like the times to cracking pressure, the times to P_{Imax} were similar among the critical care ventilators, ranging

Table 2. Cracking Pressure, Time to Cracking Pressure, Maximum Negative Inspiratory Pressure, and Time to Maximum Negative Inspiratory Pressure During Simulated Spontaneous Breathing* with Failure of 9 Portable Ventilators

Ventilator or Endotracheal Tube	Cracking Pressure (cm H_2O)	Time to Cracking Pressure (ms)	Maximum Negative Inspiratory Pressure (cm H_2O)	Time to Maximum Negative Inspiratory Pressure (ms)
8.0 mm ID endotracheal tube only	−0.04 ± 0	40 ± 0	−0.06 ± 0.03	270 ± 0
Generic single-limb circuit	−0.76 ± 0	110 ± 0	−1.31 ± 0	330 ± 0
Achieva	−1.78 ± 0.02	110 ± 0	−5.38 ± 0.03	466 ± 13.42
Avian	−3.41 ± 0.03	110 ± 5.48	−9.13 ± 0.02	456 ± 17.89
HT 50	−2.58 ± 0.02	124 ± 19.49	−6.04 ± 0.08	440 ± 5.48
LP 10	−1.99 ± 0.02	116 ± 5.48	−5.61 ± 0	478 ± 10.95
LTV 1000	−1.55 ± 0.03	78 ± 4.47	−4.83 ± 0	454 ± 5.48
Oxylog 2000	−9.56 ± 0.04	168 ± 8.37	−17.05 ± 0.06	378 ± 16.43
Tbird	−2.84 ± 0.02	100 ± 4.47	−13.77 ± 0.02	406 ± 5.48
Univent 750	−5.17 ± 0.03	146 ± 5.48	−11.73 ± 0.04	408 ± 4.47
Univent 754	−4.19 ± 0.03	108 ± 4.47	−9.89 ± 0.06	416 ± 8.94

*Tidal volume 0.5 L, peak inspiratory flow 1 L/s, respiratory rate 12 breaths/min.
ID = internal diameter.
Values are mean ± SD.

Table 3. Imposed Work of Breathing, and Imposed Pressure-Time Product During Simulated Spontaneous Breathing* with Failure of 8 Critical Care Ventilators

	Imposed Work of Breathing (mJ/L)	Imposed Pressure-Time Product (cm H_2O · s/L)
8.0 mm ID endotracheal tube only	44.96 ± 0.14	0.81 ± 0.01
2-limb circuit and 8.0 mm ID endotracheal tube only	59.36 ± 0.36	0.94 ± 0.02
7200ae	213.07 ± 3.53	2.67 ± 0.01
8400st	436.76 ± 2.23	5.50 ± 0.03
Amadeus	589.69 ± 2.71	7.42 ± 0.70
Evita 4	341.89 ± 2.26	4.14 ± 0.02
Galileo	526.59 ± 1.66	6.70 ± 0.02
Siemens 300	249.76 ± 0.62	3.13 ± 0.02
Tbird	890.63 ± 0.88	13.37 ± 0.01
Veolar	416.64 ± 0.40	5.18 ± 0.01

*Tidal volume 0.5 L, peak inspiratory flow 1 L/s, respiratory rate 12 breaths/min.
ID = internal diameter.
Values are mean ± SD.

from 406 ms with the Tbird to 466 ms with the 8400st and Siemens 300. The lowest WOB_I (213.07 mJ/L) and PTP_I (2.67 cm H_2O · s/L) were with the 7200ae. The highest WOB_I (890.63 mJ/L) and PTP_I (13.37, cm H_2O · s/L) were with the Tbird.

There was an even wider range of measured values with the portable ventilators. Among the portable ventilators the least negative cracking pressure (–1.55 cm H_2O) and time to cracking pressure (78 ms) were with the LTV 1000. The most negative cracking pressure (–9.36 cm H_2O) and time to cracking pressure (168 ms) were with the Oxylog 2000. The least negative P_{tmax} (–4.83 cm H_2O) was with the LTV 1000. The most negative P_{tmax} (–17.05 cm H_2O) was with the Oxylog 2000. The shortest time to P_{tmax} (378 ms) was with the Oxylog 2000. The longest time to P_{tmax} (478 ms) was with the LP 10. The lowest WOB_I (361.37 mJ/L) and PTP_I (4.52 cm H_2O · s/L) were with the LTV 1000. The highest WOB_I (969.60 mJ/L) and PTP_I (16.70 cm H_2O · s/L) were with the Oxylog 2000.

Discussion

Our data indicate large differences in the measured values between the ventilators and between the groups of ventilators. For the critical care ventilators, based on the cracking pressure, P_{tmax}, and times to those pressures, it is not surprising that the lowest WOB_I (213.07 mJ/L) and PTP_I (2.67 cm H_2O · s/L) were with the 7200ae and that the highest WOB_I (890.63 mJ/L) and PTP_I (13.37 cm H_2O · s/L) were with the Tbird. Among the portable ventilators the cracking pressure, P_{tmax}, and times to those pressures are congruent with the results that the lowest WOB_I (361.37 mJ/L) and PTP_I (4.52 cm H_2O · s/L) were measured with the LTV 1000, and the highest WOB_I (969.60 mJ/L) and PTP_I (16.70 cm H_2O · s/L) were measured with the Oxylog 2000.

The pressure-volume loops suggest that the anti-asphyxia valves on some of the critical care ventilators operate in a similar fashion (7200ae and Siemens 300, and 8400st and Veolar). In addition, the pressure-volume loops suggest that the anti-asphyxia valves in ventilators such as the 7200ae and Siemens 300 open at lower cracking pressures and have lower resistance to flow, allowing for lower P_{tmax},

Table 4. Imposed Work of Breathing, and Imposed Pressure-Time Product During Simulated Spontaneous Breathing* with Failure of 9 Portable Ventilators

	Imposed Work of Breathing (mJ/L)	Imposed Pressure-Time Product (cm H_2O · s/L)
8.0 mm ID endotracheal tube only	44.96 ± 0.14	0.81 ± 0.01
Generic single-limb circuit and 8.0 mm ID endotracheal tube only	96.59 ± 0.03	1.32 ± 0.01
Achieva	388.02 ± 0.77	4.72 ± 0.01
Avian	644.14 ± 56.30	8.50 ± 0.74
HT 50	447.00 ± 3.27	5.63 ± 0.05
LP 10	403.98 ± 0.48	4.92 ± 0.01
LTV 1000	361.37 ± 1.22	4.52 ± 0.01
Oxylog 2000	969.60 ± 22.70	16.70 ± 0.37
Tbird	890.63 ± 0.88	13.37 ± 0.01
Univent 750	782.84 ± 1.53	11.04 ± 0.02
Univent 754	719.22 ± 1.97	9.57 ± 0.01

*Tidal volume 0.5 L, peak inspiratory flow 1 L/s, respiratory rate 12 breaths/min.
ID = internal diameter
Values are mean ± SD

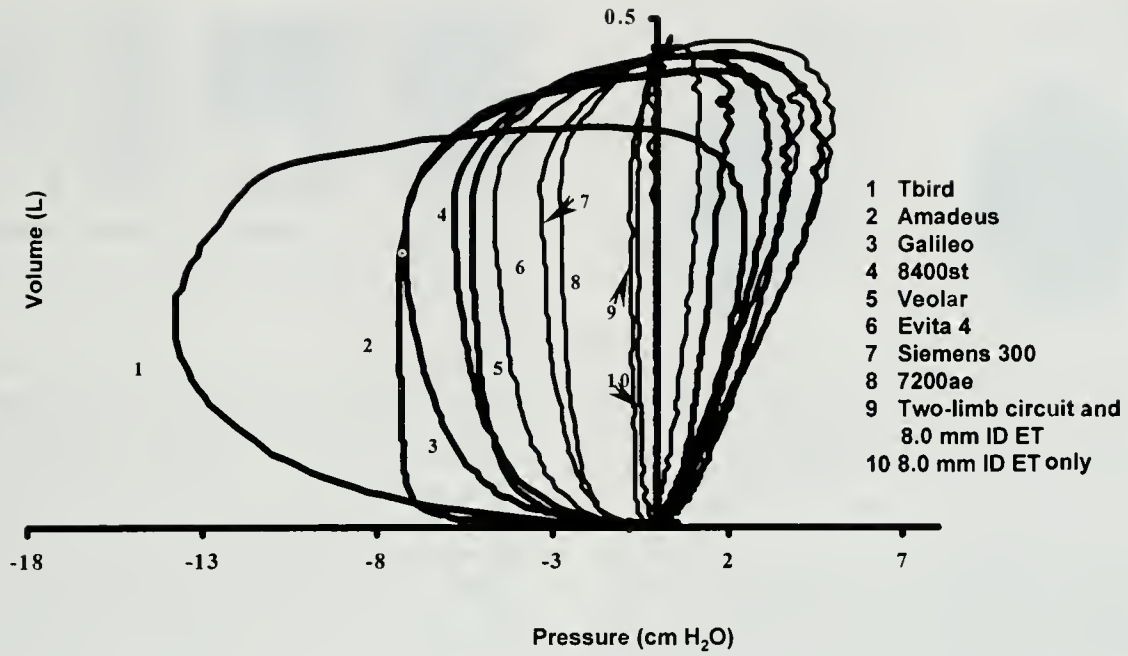

Fig. 1. Pressure-volume loops from simulated spontaneous breathing (tidal volume 0.5 L, peak inspiratory flow 1 L/s, rate 12 breaths/min) during failure of 8 critical care ventilators. See text for descriptions and manufacturers of ventilators. ID = internal diameter. ET = endotracheal tube.

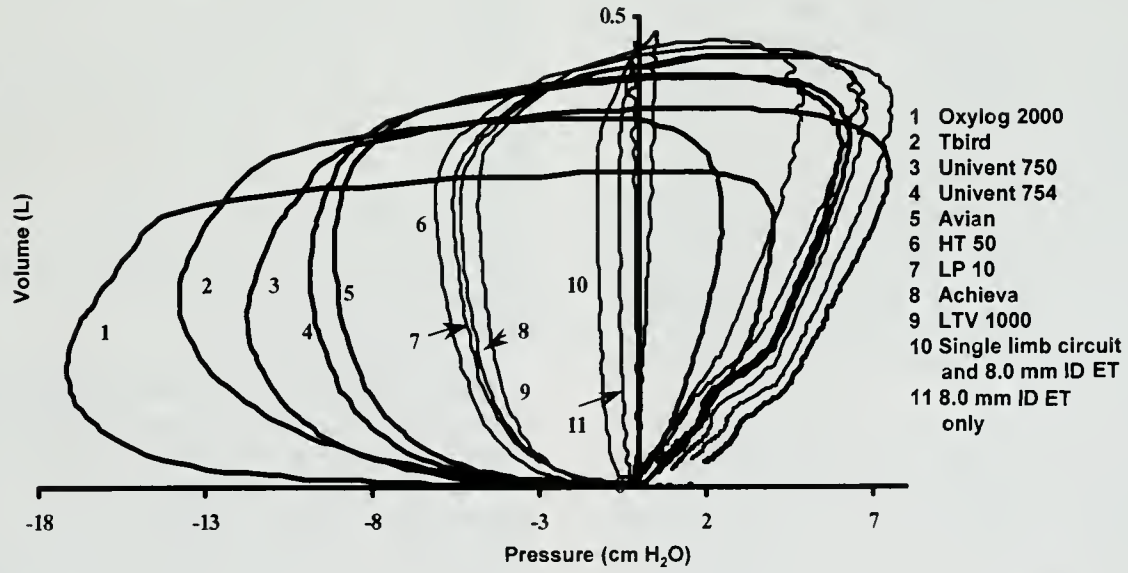

Fig. 2. Pressure-volume loops from simulated spontaneous breathing (tidal volume 0.5 L, peak inspiratory flow 1 L/s, rate 12 breaths/min) during failure of 9 portable ventilators. See text for descriptions and manufacturers of ventilators. ID = internal diameter. ET = endotracheal tube.

WOB_I, and PTP_I. Ventilators such as the Tbird have anti-asphyxia valves that open at lower cracking pressures but create more resistance to flow, resulting in a higher P_{Imax}, WOB_I, and PTP_I. The anti-asphyxia valve in the Amadeus opened at a higher cracking pressure but once opened had lower resistance to flow, resulting in a lower WOB_I and PTP_I than the Tbird.

The pressure-volume loops for the portable ventilators suggest that the anti-asphyxia valves on the HT 50, LP 10, Achieva, and LTV 1000 operate in a similar fashion. The pressure-volume loops also suggest that, in a progressive manner, the anti-asphyxia valves on the Avian, Univent 754, Univent 750, Tbird, and Oxylog 2000 create more resistance to flow, resulting in greater WOB_I and PTP_I.

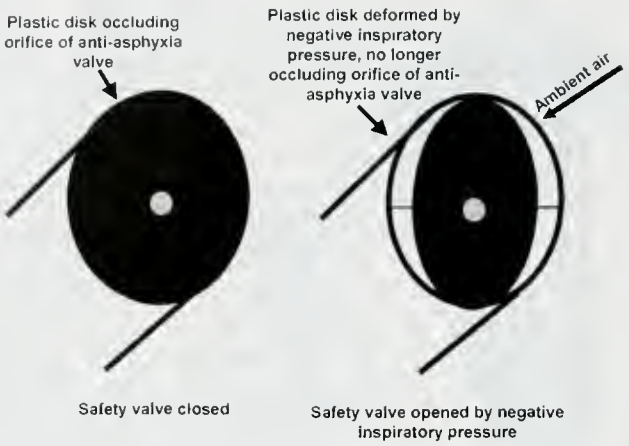
Plastic disk occluding orifice of anti-asphyxia valve
Plastic disk deformed by negative inspiratory pressure, no longer occluding orifice of anti-asphyxia valve
Ambient air
Safety valve closed
Safety valve opened by negative inspiratory pressure

Fig. 3. Schematic drawing of typical anti-asphyxia valve.

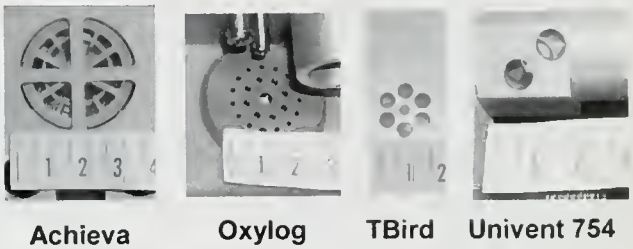

Fig. 4. Inlets of anti-asphyxia valves from various ventilators (units are centimeters).

The WOB_I during ventilator failure is dependent on the design of the anti-asphyxia valve and pathway of the ambient air entrained through the valve, as governed by Poiseuille's law.[21] Though with some ventilators the anti-asphyxia valves can be examined without disassembling the ventilator, many require disassembly to examine the valve as well as the gas pathway.

The anti-asphyxia valves are typically simple one-way valves, which are (usually plastic) disks that normally occlude an orifice on the ventilator's inspiratory limb. When sufficient negative pressure occurs in the inspiratory limb the plastic disk deforms and allows ambient air to be entrained into the inspiratory limb (Fig. 3).

The initiation of air flow through the anti-asphyxia valve is determined by the cracking pressure and the time it takes to reach the cracking pressure.[13] Air flow through the valve is also determined by the time it takes the valve to fully open (reflected by the time to P_{Imax}) and the resistance across the fully open valve (reflected by P_{Imax}). Figure 4 illustrates the different surface areas of the inlet of 4 anti-asphyxia valves.

The engineering challenge is to design a valve that does not open at low inspiratory pressure, because that would allow entrainment of ambient air into the oxygen-enriched breathing mixture and thus lower the fraction of inspired oxygen (F_{IO_2}). However, if the resistance across the open valve is too great or the valve takes a long time to open, it will impose more work, resulting in patient discomfort and respiratory muscle fatigue.

The ASTM standard (F1246) for home care ventilators calls for the cracking pressure of unidirectional anti-asphyxia valves to be no less than 3 and no greater than 10 cm H_2O.[13] In addition, the resistance to flow across the valve is not to exceed 10 cm $H_2O \cdot s/L$ when a spontaneous breath is generated at the lowest and highest recommended V_T. The ASTM standard (F1100) for critical care ventilators calls for a unidirectional inspiratory relief valve that opens when the patient inspires spontaneously and the flow of inspiratory gas from the ventilator is inadequate. The standard also calls for the resistance across the valve to be measured at a constant flow of 30 L/min.[12]

The results of the present investigation suggest the anti-asphyxia valves on some of the tested ventilators could open prematurely (ie, below 3 cm H_2O) under the test conditions. Specifically, all of the critical care ventilators, except for the Amadeus and some of the portable ventilators (Achieva, HT 50, LP 10, LTV 1000, and Tbird), could open prematurely. The risk from premature valve opening is reduced F_{IO_2} in the inspired gas. However, if the ventilator is triggered fast enough and it provides sufficient inspiratory flow during spontaneous breathing, the anti-asphyxia valve should not open.

Banner et al suggest that WOB_I plus physiologic WOB make up the total WOB.[22] Normal physiologic WOB is reported to be between 300 and 500 mJ/L.[23] All the critical care ventilators imposed a WOB of at least half of normal WOB, except for the 7200ae and Siemens 300. All of the portable ventilators imposed a WOB of at least half of normal WOB. That imposed work may lead to patient discomfort and respiratory muscle fatigue.[24] However, imposed work is not an issue if the patient is not making spontaneous inspiratory efforts.

An extensive literature search failed to reveal any prior investigations of WOB_I during ventilator failure. During proper ventilator functioning, investigators found that critical care ventilators impose little work (< 40 mJ/L) when operating in spontaneous breathing modes.[1,2,4,6,7] The WOB_I of earlier portable ventilators, lacking a demand valve, was in the range of 133–215 mJ/L, leading to recommendations of retrofitting those devices with a demand valve if they are used with spontaneously breathing patients.[5,8] Later models, with an integral demand valve, imposed a lower WOB (4–121 mJ/L) when tested under conditions similar to those used in this study.[8,9]

The present study has several limitations. Advantages of using a lung model include accuracy and precision of creating the test conditions and no threat to the health of human subjects. Since the investigator does not have to wait for the availability of volunteers, this design is economical in terms of time. However, this design also has a

distinct drawback in terms of external validity and generalizability to the population of interest (critically ill patients requiring mechanical ventilation). There are numerous potentially intervening variables in human subjects that are not present in a lung model, including the effect of airway moisture on the functioning of the ventilator and the effect of varying V_T, peak inspiratory flow, and respiratory rate. In addition, our model used a constant-flow breathing pattern, but a sinusoidal pattern would have better simulated spontaneous breathing. Also our model gives no information on patient perception of discomfort while breathing through a failed ventilator. To represent clinical conditions, ventilators were selected from those awaiting patient use. Per the manufacturer's instructions, preventive maintenance had been conducted on the ventilators at the prescribed intervals, and the ventilators were calibrated and prepared per the manufacturer's specifications. The use history of the ventilators used in this investigation was not recorded, and we recognize that differences in ventilator use history could affect the functioning of the anti-asphyxia valve.

Future studies should vary the V_T, peak inspiratory flow, and respiratory rate and should include more than one of each ventilator model, to better represent the various models of these devices. In addition, the possibility of premature opening of the anti-asphyxia valve should be investigated during normal operation. The study should be repeated using nonintubated, healthy human subjects.

Conclusions

Spontaneous breathing through the anti-asphyxia valve during ventilator failure may impose work approximating the physiologic work of breathing, thus effectively doubling total work of breathing and preventing effective breathing, which may result in respiratory muscle fatigue. Also, some anti-asphyxia valves have the potential to open prematurely during spontaneous breathing with normal ventilator function and thus threaten to reduce the F_{IO_2}. Manufacturers should examine the functioning of anti-asphyxia valves in existing and future ventilators. Our results reinforce the need to properly monitor mechanically ventilated patients and to have sufficient back-up power supplies and a method of manual ventilation.

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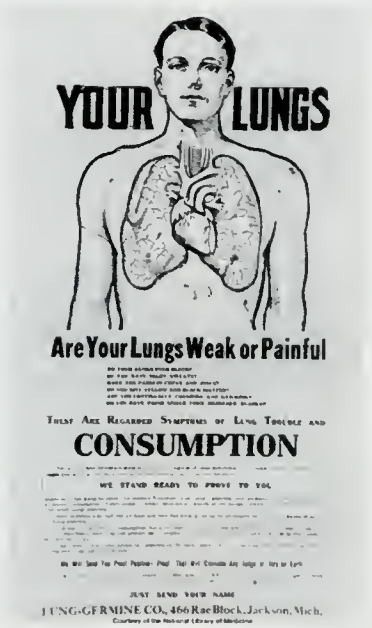
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Noninvasive Nocturnal Ventilatory Support in Advanced Lung Disease from Cystic Fibrosis

John T Granton MD, Colin Shapiro MD, and Steven Kesten MD

INTRODUCTION: Respiratory failure is the most common cause of death among people with cystic fibrosis (CF). The role of noninvasive positive-pressure ventilation (NPPV) in severe but stable lung disease from CF has not been adequately determined. We therefore sought to determine the tolerability and efficacy of NPPV in people with advanced lung disease from CF. METHODS: We studied 13 patients with CF, severe air flow limitation (mean ± SD forced expiratory volume in the first second 0.8 ± 0.3 L), and chronic respiratory failure (mean ± SD P_{aO_2} 67 ± 19 mm Hg and P_{aCO_2} 51 ± 8 mm Hg). All patients were awaiting lung transplantation. NPPV was prescribed for 2 months. Overnight polysomnography, arterial blood gas values, pulmonary function, exercise capacity, and health-related quality of life were evaluated at baseline, after 2 months of NPPV, and again after a 1-month wash-out period off NPPV. RESULTS: Eight patients completed the study and tolerated NPPV extremely well. These patients subjectively noted improvement in their symptoms and requested that they restart NPPV after completing the protocol. Nevertheless, NPPV did not improve objective measures of sleep quality, daytime blood gas values, pulmonary function, respiratory muscle strength, or exercise tolerance. All 8 patients were continued on NPPV and underwent successful lung transplantation. CONCLUSIONS: NPPV is well tolerated by most patients with CF and severe lung disease and may be a useful bridge to support CF patients awaiting lung transplantation. However, despite subjective improvement, standard objective measures did not improve and cannot explain the reduced symptoms reported by these patients. *Key words: cystic fibrosis, noninvasive positive pressure ventilation, NPPV, bi-level positive airway pressure, BiPAP, sleep.* [Respir Care 2002;47(6):675–681]

Introduction

Noninvasive positive-pressure ventilation (NPPV) has been successfully used in patients with chronic respiratory failure secondary to neuromuscular and skeletal disorders.[1] In patients with acute respiratory failure (ARF) from chronic obstructive pulmonary disease (COPD), NPPV reduces the requirement for intubation and mechanical ventilation and may reduce mortality.[2] Experience with NPPV in COPD patients with stable hypercapnic respiratory failure is evolving and was recently extensively reviewed.[3] When combined with domiciliary oxygen, NPPV improves daytime blood gas values and quality of life more than oxygen alone.[4,5] However, other studies have not shown more benefit from NPPV than from sham ventilation[6] or oxygen therapy alone.[7] Respiratory failure is responsible for the majority of morbidity and mortality in adult cystic fibrosis (CF) patients. Previous studies evaluated the role of NPPV in patients with acute exacerbations of CF. Padman et al reported their experience with NPPV in 15 pediatric patients with ARF (4 with CF).[8] All 4 CF patients were supported through ARF and were maintained on NPPV for 3 weeks to 1.5 years until lung transplantation. Two centers have specifically evaluated the use of NPPV in (a total of 14) CF patients. Both studies found initial improvement in gas exchange and that NPPV could be tolerated for periods of up to 18 months.[9,10] Hill et al reported that their patients had subjective improvement in sleep, measurable increase in forced vital capacity, and reduced P_{aCO_2}.[10] Un-

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fortunately, no formal comparative assessments of sleep, exercise, or quality of life were made before and after NPPV. In addition, those patients were studied during documented deterioration.

Less information is available on the efficacy of NPPV in patients with stable CF. We recently found that NPPV acutely improved gas exchange and breathing pattern in patients with chronic respiratory failure and stable lung function.[11] We also observed that NPPV reduced pleural pressure swings during tidal breathing and concluded that NPPV might reduce patients' effort of breathing. Thus, NPPV is a potentially useful modality to manage CF patients with chronic respiratory failure. We postulated that NPPV would improve sleep architecture, daytime blood gas values, exercise tolerance, and health-related quality of life in patients with advanced CF awaiting lung transplantation. Chronic respiratory failure is the leading cause of death in people with CF, and among those who are appropriate candidates for lung transplantation, many die because of donor shortages and the increasing waiting times to transplantation. Based on experience with NPPV in patients with acute respiratory exacerbations of CF, NPPV may be useful in patients awaiting lung transplantation.

Methods

Study Population

Patients with CF referred to The Toronto Hospital and accepted for lung transplantation were considered for enrollment into the study. Subjects were considered eligible if they were over the age of 18 years and had $P_{aO_2} < 50$ mm Hg or $P_{aCO_2} > 40$ mm Hg. All the patients were clinically stable and had been free from acute exacerbation of respiratory disease requiring hospitalization in the preceding month. Patients with active infection, history of hemoptysis, or recent pneumothorax were excluded. The protocol was approved by The Toronto Hospital Committee for Research on Human Subjects. All participants provided informed consent prior to enrollment in the study.

Study Protocol

Following the completion of baseline measurements and education about nasal NPPV, NPPV was administered nightly for a 2-month period. NPPV was provided using bi-level positive airway pressure (BiPAP, Respironics, Monroeville, Pennsylvania) applied through a nasal mask. All NPPV breaths were patient-initiated, using the spontaneous or "S" mode. To optimize the likelihood of patient acceptance and tolerance, the levels of inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP) were determined by titrating levels to patient comfort prior to the study. The acute effects of this level of NPPV in this population was reported previously and seemed to decrease P_{aCO_2}, increase tidal volume (V_T), and reduce respiratory rate, and there was a trend toward reduced esophageal pressure swings and diaphragmatic electromyographic activity.[8] No further attempt was made to adjust the pressure levels during the study. Patients were instructed to use NPPV while sleeping during a 2–3 day hospitalization period and were discouraged from using NPPV during the daytime. We specifically wanted to avoid using NPPV as a long-term ventilation mode and to maintain patient independence so as not to interfere with daytime activities. Pulmonary function, respiratory muscle strength, exercise testing, sleep, and quality of life were measured at baseline, after 2 months of NPPV, and again following a 1-month period off NPPV. Patients were followed at 1-month intervals in the clinic. No other routine follow-up was provided.

Sleep Study

Each participant underwent 3 formal overnight polysomnographic evaluations. Only the second sleep evaluation was performed while the patient was receiving NPPV. A respiratory inductance plethysmograph (Respitrace, Ambulatory Monitoring, White Plains, New York), calibrated by the volume of isoflow method, was used to measure V_T and excursion of the rib cage and abdomen. Electrocardiogram recordings were made with a single precordial lead. Arterial oxyhemoglobin saturation (S_{pO_2}) was measured with an ear oximeter (Oxyshuttle, SensorMedics/VIASYS Healthcare, Yorba Linda, California). Mean low S_{pO_2} during sleep was determined by averaging the lowest S_{pO_2} every 30 seconds throughout the night. The amount of time spent in sleep stages 1, 2, 3, and 4, and rapid eye movement (REM) was recorded. Total sleep time was defined as the amount of time spent in any stage of sleep relative to the total recording time.

Pulmonary Function and Respiratory Muscle Strength

To determine if the effects of NPPV carried through the daytime, resting arterial blood gas (ABG) values were measured at noon. Spirometry,[12] maximum voluntary ventilation, and lung volumes via body plethysmography were assessed for each interval. Maximum static inspiratory pressure at residual volume and maximum static expiratory pressure at total lung capacity were used as indices of respiratory muscle strength. For these measurements the patient breathed through a mouth piece attached to a 3-way valve connected to a wedge spirometer (Model 922, Sensormedics, Yorba Linda, California). Mouth pressures were measured with a pressure transducer (MP 45, Validyne, Northridge, California). Measurements were performed un-

til 3 recordings varied < 10% and were sustained for 1 second.

Exercise Testing

Maximal exercise testing was performed on a treadmill (T500, Burdick, Milton, Wisconsin), with continuous monitoring of respiratory gases (Ametek, AEI Technologies, Pittsburgh, Pennsylvania), S_{pO_2} (Invivo 4500, Orlando, Florida), and heart rate (MSC 2001, Medical Systems International, Greenvale, New York). Work rate was increased at 2-min intervals, according to the perceived fitness of the study subject. Subjects were encouraged to exercise to a symptom-limited maximum level. The maximum values were recorded for the last minute of exercise and included oxygen consumption, peak oxygen consumption, maximum voluntary ventilation, and heart rate. Also recorded were maximum work load, duration of exercise, and level of blood oxygen desaturation during exercise.

Quality of Life Assessment

Symptoms and quality of life were measured with the Chronic Respiratory Disease Questionnaire (CRQ) and the Asthma Quality of Life Questionnaire (AQLQ). Both questionnaires reflect impairments perceived to be important by the patient and are sensitive to changes that develop over a 2-week period.[13–15]

The CRQ has been validated for patients with chronic respiratory failure. It evaluates 4 domains: dyspnea (5 items), fatigue (4 items), emotional function (7 items), and disease mastery (4 items).

The AQLQ measures quality of life in asthma patients. It evaluates 4 domains: activity limitations (11 items), symptoms (12 items), emotional function (5 items), and exposure to environmental stimuli (4 items).

For both the AQLQ and CRQ each item in each domain is equally weighted. The possible responses for each item lie along a 7-point intensity scale in which 1 indicates maximum impairment and 7 indicates no impairment. The final score is expressed as the average response for each domain, and overall quality of life is expressed as the average of all the domains. With both questionnaires, an average change in response of 0.5 in any domain is considered to represent a meaningful difference.[16]

Data Analysis

Data are presented as either mean ± SD or as proportions. Comparison of the 3 time periods was made using the 2-tailed, paired Student's *t* test. Differences were considered statistically significant if p was < 0.05.

Table 1. Polysomnography Measurements

	Baseline	After 2 Months of NPPV	After 1-Month Wash-out Period
Efficiency (% TST)	76 ± 15	82 ± 8	85 ± 11
Latency (min)	21 ± 38	23 ± 26	16 ± 19
Stages 1 + 2 (% TST)	40 ± 10	45 ± 8	33 ± 16
Stages 3 + 4 (% TST)	26 ± 8	28 ± 9	30 ± 16
REM sleep (% TST)	13 ± 5	13 ± 3	16 ± 5
Awakenings (*n*)	34 ± 17	45 ± 25	31 ± 23
Mean low S_{pO_2} (%)	86 ± 9	87 ± 5	86 ± 5
Mean high P_{tcCO_2} (mm Hg)	64 ± 13	60 ± 8	60 ± 13

NPPV = noninvasive positive-pressure ventilation
TST = total sleep time
REM = rapid eye movement
S_{pO_2} = oxygen saturation measured via ear oximetry
P_{tcCO_2} = partial pressure of carbon dioxide measured transcutaneously
Values are mean ± SD

Results

Thirteen patients (9 men, 4 women, 24–37 years of age) with CF and severe air flow limitation (P_{aO_2} of 67 ± 19 mm Hg and P_{aCO_2} of 51 ± 8 mm Hg) were evaluated. Eight patients had been reported on in our earlier study of the acute effects of NPPV.[11] Eight of the 13 enrolled patients completed the 2 months of NPPV and the 1-month wash-out period. Five patients did not complete the study, for the following reasons: 1 developed hemoptysis, 1 had a poor-fitting mask, 1 received a lung transplant, 1 refused to discontinue NPPV after 2 months, and 1 refused full participation with the study protocol. The 8 patients reported herein did not require changes to their masks once they had been fitted on the initial visit. All patients subjectively noted improvement in symptoms and requested to restart NPPV after the completion of the protocol. The patients who completed the protocol continued on NPPV and survived to transplantation.

Sleep Study

All patients who completed the study had full sleep assessments. During NPPV there were no significant changes in sleep efficiency, latency, or sleep stages (Table 1). There was a nonsignificant increase in awakenings and a nonsignificant decrease in mean high transcutaneously-measured P_{CO_2}, from 64 ± 13 mm Hg to 60 ± 8 mm Hg. There was no change in mean low S_{pO_2} during the study.

Pulmonary Function Testing

One patient failed to complete the final pulmonary function assessment. Pulmonary function test results did not

Table 2. Physiologic Measurements

	Baseline	After 2 Months of NPPV	After 1-Month Wash-out Period
P_{aO_2} (mm Hg)	67 ± 19	69 ± 13	64 ± 14
P_{aCO_2} (mm Hg)	51 ± 8	50 ± 4	47 ± 7
FEV_1 (L)	0.8 ± 0.3	0.8 ± 0.3	0.9 ± 0.4
FVC (L)	1.7 ± 0.6	1.7 ± 0.5	1.8 ± 0.7
MIP (% of predicted)	67 ± 25	78 ± 25	78 ± 28
MEP (% of predicted)	56 ± 17	71 ± 20	70 ± 16
MVV (L/min)	40 ± 26	39 ± 20	43 ± 24

FEV_1 = forced expiratory volume in the first second
FVC = forced vital capacity
MIP = maximum inspiratory pressure
MEP = maximum expiratory pressure
MVV = maximum voluntary ventilation
Values are mean ± SD

change during the study. There were no significant changes in FEV_1, forced vital capacity, total lung capacity, or residual volume (Table 2). There was a nonsignificant increase in maximum static inspiratory pressure, from 67 ± 25% to 78 ± 25% of predicted, and a nonsignificant increase in maximum static expiratory pressure, from 56 ± 17% to 71 ± 20% of predicted. The estimates of respiratory muscle strength did not return to baseline values following the 1-month wash-out period (see Table 2). Blood gas values were incomplete in 4 patients. In the remainder there was no significant change in P_{aO_2}, but there was a nonsignificant trend toward a decrease in P_{aCO_2} over the 3-month study (see Table 1).

Exercise Studies

Because of respiratory disability and requirement for supplemental oxygen, only 4 patients were able to perform stage 1 exercise testing. In these patients there was no significant improvement in peak oxygen consumption, maximum voluntary ventilation, exercise duration, or work load (Table 3). Similarly, there was no change in the degree of arterial oxygen desaturation or maximum heart rate.

Quality of Life Assessments

During the study there was a trend toward a gradual improvement in the quality of life in all categories in both the CRQ and AQLQ (Table 4). Unfortunately, the information from 1 patient was incomplete for the 2-month questionnaire and from 2 patients for the wash-out questionnaire. The total score for the CRQ at baseline was 3.7 ± 1, which increased to 4.0 ± 0.8. During the wash-out phase the CRQ score increased further, to 4.4 ± 0.3.

Table 3. Graded Exercise Testing Measurements

	Baseline	After 2 Months of NPPV	After 1-Month Wash-out Period
Peak V_{O_2} (L/min)	38 ± 13	37 ± 12	39 ± 10
Duration (min)	12 ± 3	12 ± 2	13 ± 2
Work (watts)	433 ± 152	382 ± 155	533 ± 231

V_{O_2} = oxygen consumption
Values are mean ± SD.

The changes in AQLQ score from baseline (3.9 ± 1) to after 2 months of NPPV (4.2 ± 1) and to the completion of the wash-out phase (4.7 ± 0.8) were not statistically significant.

With regard to individual CRQ scores, after 2 months of NPPV meaningful improvement (ie, > 0.5 increase for any domain) had occurred in 3 patients in the dyspnea domain, 6 patients in the fatigue domain, 3 patients in the emotional function domain, and 3 patients in the disease mastery domain. These improvements were sustained throughout the wash-out period.

Two patients did not change significantly in any CRQ domain during the study, and 1 patient had a deterioration > 0.5 in the fatigue and emotional function domains following 2 months of NPPV, which coincided with an acute infectious exacerbation.

With regard to individual AQLQ scores, after 2 months of NPPV meaningful improvement (ie, > 0.5 increase for any domain) had occurred in 3 patients in the activity level domain, in 5 patients in the symptom domain, and in 1 patient in the environmental domain. One patient had de-

Table 4. Quality of Life Measurements

Symptom Score Domain	Baseline	After 2 Months of NPPV	After 1-Month Wash-out Period
Chronic Respiratory Failure Questionnaire			
Dyspnea	3.0 ± 0.7	3.6 ± 0.8	3.5 ± 0.7
Emotional	3.4 ± 1.5	3.5 ± 1.0	4.2 ± 0.7
Fatigue	4.1 ± 1.4	4.2 ± 1.0	4.9 ± 0.4
Mastery	4.4 ± 1.4	4.6 ± 1.2	5.1 ± 0.6
Total Scores	3.7 ± 1.0	4.0 ± 0.8	4.4 ± 0.3
Asthma Quality of Life Questionnaire			
Activity	3.8 ± 1	4.1 ± 1	4.5 ± 1
Symptoms	3.5 ± 1	4.0 ± 1	3.9 ± 1
Emotional	4.5 ± 2	4.4 ± 2	4.9 ± 1
Environmental	4.9 ± 1	4.9 ± 1	5.3 ± 1
Mean Score	3.9 ± 1	4.2 ± 1	4.7 ± 0.8

Values are mean ± SD

terioration (ie, > 0.5 decrease) in the emotional function domain after 2 months of NPPV. Three other patients had improvement in emotional function domain after the wash-out period.

No estimates were made of patient compliance with NPPV. All 8 patients reported that they were tolerating the NPPV at the prescribed settings at 2 months. After the wash-out phase, all 8 patients continued NPPV until lung transplantation.

Discussion

This is the first prospective study of NPPV in outpatients with CF and advanced respiratory disease. With the NPPV settings we employed there was no improvement in daytime ABG values, spirometry, respiratory muscle strength, exercise tolerance, or sleep architecture.

Previous reports have evaluated NPPV for patients with deteriorating lung function. In the largest study to date, Hill et al reported their 5-year experience in 10 hospitalized patients with hypercapnic respiratory failure (P_{aCO_2} 65.4 ± 4.5 mm Hg). Two did not tolerate NPPV, 5 experienced a quantifiable improvement in P_{aCO_2} after 3 months of NPPV, and 10 had an increase in forced vital capacity by 2 months of NPPV.[10] They also reported a subjective improvement in sleep and morning headache. No formal assessments of sleep or symptom severity were made.

The subjects in our study all had severe air flow limitation (FEV_1 0.8 ± 0.3 L) and hypercapnia (resting P_{aCO_2} 51 ± 8 mm Hg), but, in contrast to previous studies, all were clinically stable outpatients. In an earlier report on the acute effects of BiPAP (including 7 of the patients reported in this study), we found that NPPV reduced respiratory rate and increased V_T.[11] Minute ventilation was also reduced, and S_{pO_2} and transcutaneously-measured P_{CO_2} significantly improved. In addition, we found that NPPV significantly reduced pleural pressure swings during quiet breathing. This suggested to us that NPPV could reduce hypoxemia, improve alveolar ventilation, and reduce the work of breathing in lung disease due to CF.

People with severe lung disease from CF often have substantial derangement of sleep quality and desaturate during sleep.[17–19] Spier et al observed lower sleep efficiency, less REM sleep, and more arousals in CF patents than in normal subjects.[18] In that study, 2 of 10 subjects with CF had concurrent obstructive sleep apnea. In addition, CF patients have large variations in nocturnal V_T, suggesting that alterations in the control of breathing may contribute to nocturnal desaturation.[19]

Our study patients had substantial impairment in sleep architecture and efficiency, reflected by the higher mean number of awakenings and less time spent in sleep stages 3 and 4. As well, our subjects had mild hypoxemia and substantial hypercapnia during sleep. No patient had more than 5 obstructive episodes per hour. With the application of NPPV there was a nonsignificant trend toward improvements in sleep efficiency and in transcutaneously-measured P_{CO_2} during sleep. There was no improvement in REM or stage 4 sleep time. Indeed, there was a nonsignificant trend toward an increase in the mean number of awakenings with NPPV, so it may have interfered with sleep in these patients.

Our negative findings contrast with a previous study's observation of improvement in the respiratory disturbance index and baseline S_{pO_2} during REM and non-REM sleep in 7 CF patients during the nocturnal application of nasal continuous positive airway pressure.[20] That study used much higher levels of end-expiratory pressure (11 ± 2 cm H_2O), which might account for the observed differences in outcome. Ideally, titration of the IPAP and EPAP levels should have been adjusted during the sleep study. However, based on the acute effects of BiPAP in these patients,[8] we believe that the potential benefit of increasing the pressure would have been offset by patient intolerance. Future studies should address this concern.

Previous studies evaluating the acute effects of NPPV for ARF in CF patients found that morning ABG values were improved following a night of NPPV.[9] In the cohort we studied we saw no improvement in oxygenation or hypercapnia. It is unclear if the difference was related to differences in the timing of ABG sampling between the 2 studies. We also found no improvement in the severity of air flow limitation or the estimates of muscle strength. We initially hypothesized that NPPV would rest the respiratory muscles and thereby improve their work efficiency through the day. However, we found no significant improvement in exercise tolerance.

Severe lung disease in CF substantially impairs quality of life and functional status.[21] Few studies have incorporated quality-of-life assessments into therapeutic trials with CF patients. Unfortunately, specific measures of lifestyle impairment in CF patients have not been developed systematically. For the purposes of this study we chose 2 questionnaires (CRQ and AQLQ) that have been validated for other respiratory disease patients.[13–15] Both CRQ and AQLQ are reliable, easy to administer, and sensitive to changes in a patient's health that occur over a period of 2 weeks.[16]

Using the CRQ and AQLQ scoring systems we found a nonsignificant trend toward improved quality of life during the duration of the study. The questionnaire scores showed continued improvement during the wash-out phase, in some patients, which suggests that there was either a carryover effect of NPPV into the wash-out phase or that there was no causal relationship between NPPV and the observed improvement. However, the trends we observed were not statistically significant, perhaps reflecting our small sample size. The lack of statistical significance con-

trasts with the patients' nonquestionnaire reporting to us of an overall sense of improved well-being and sleep. However, we cannot exclude the possibility that the patients' responses may have reflected a desire to please their caregivers. In addition, the questionnaires we used have not been validated in CF patients, so they may be insensitive to symptoms or sense of well-being in these patients.

We were disappointed by the lack of objective improvement during this study. The results are similar to earlier observations in patients with ARF from a variety of causes, which suggested that NPPV might not be useful for patients with bronchiectasis.[22]

There are several possible explanations for our observations. The hypothesis that NPPV will benefit CF patients may be wrong. Alternatively, our patient selection may have been incorrect. For COPD patients it has been recommended that only patients with substantial hypercapnia benefit from and should be considered for NPPV. However, the relationship between the degree of carbon dioxide retention and success with NPPV is controversial and remains speculative.[6,7]

Another possible explanation for our findings relates to potential weaknesses in our methodology. First, there may have been some carbon dioxide rebreathing with the NPPV system and settings employed. Ferguson and Gilmartin observed that EPAP of at least 6 cm H_2O was required to eliminate rebreathing of carbon dioxide from the tubing, using the BiPAP system, when carbon dioxide is not completely removed through the exhalation port (Whisper Swivel, Respironics, Pittsburgh, Pennsylvania).[23] They also found that that EPAP level significantly reduced the effect of rebreathing. Our patients used EPAP of 4–6 cm H_2O, making the likelihood of rebreathing remote.

Second, relatively low inspiratory support pressures were used in our study. IPAP ranged from 10 to 12 cm H_2O, which resulted in a average driving pressure of only 6 cm H_2O. Ambrosino et al evaluated the physiologic effects of 2 hours of NPPV without EPAP (using either BiPAP or pressure support by a standard ventilator) in 7 patients with stable COPD.[24] IPAP was titrated to achieve V_T of 10–15 mL/kg and, consequently, greater levels of IPAP were used (17–20 cm H_2O). They noted a significant decrease in P_{aCO_2}, from 60 to 49 mm Hg over a 12-hour period. Similar results were obtained by Brochard et al in their study of ARF in COPD patients, in which 15–20 cm H_2O pressure was applied via face mask, using a modified pressure support system.[25] Our patients had substantial air flow limitation and might have benefited from higher IPAP levels.

Third, CF patients experience frequent and rapid fluctuations in health and functional status, owing to repetitive infectious exacerbations, which may have confounded the observations of our relatively short study, because beneficial effects may have been obscured by the patients' health variations. Two months is probably sufficient to observe physiologic and sleep-related changes, but the duration of the trial may have been too short, with a longer period required before an effect could be seen.

Some of our conclusions are also hampered by the study design. We used a wash-out period to judge the effects of BiPAP and to control for variations in patient's health status. It is possible that a carry-over effect of NPPV through the wash-out period may have obscured any effects of NPPV. It is also possible that the chosen outcome measures may be too insensitive to physiologic change. We also did not use any formal method to determine compliance with NPPV. Patients may have simply not have been using NPPV as prescribed, but we think this is highly unlikely, as all patients requested to continue treatment beyond the study period. It is interesting to speculate that a subjective benefit may have occurred, given that these patients requested to continue NPPV.

Conclusions

For patients with stable CF, NPPV did not significantly improve objective measurements of physiology or quality of life. However, limitations in study design and implementation of NPPV preclude any definite conclusions regarding efficacy in this patient population, and some of the observations and trends in this trial suggest that a longer trial is needed to more fully evaluate the efficacy of NPPV in patients with chronic respiratory failure from CF, incorporating lessons learned from the limitations of the present study.

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Clinical Validation of a Continuous Intravascular Neonatal Blood Gas Sensor Introduced Through an Umbilical Artery Catheter

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INTRODUCTION: Arterial blood gas (ABG) values are a necessary diagnostic measurement in the management of critically ill neonates. We hypothesized that a fiberoptic intravascular blood gas sensor, adapted for use through an umbilical artery catheter, would produce blood gas results with clinically acceptable bias and precision, in comparison to laboratory values, but with no blood loss. METHODS: We evaluated a fiberoptic intravascular blood gas sensor (Neotrend) in 23 consecutive neonates suffering respiratory failure. The sensor was inserted into the descending aorta via a 4.0 or 4.5 French umbilical artery catheter and extended 20 mm beyond the catheter tip. Arterial blood samples were drawn as clinically indicated and analyzed using a standard laboratory analyzer. Sensor measurements were recorded at the time of arterial blood sampling. Additionally, we recorded and evaluated data related to umbilical artery catheter placement (low position [L3–L4] vs high position [T8–T10]), duration of sensor functioning and use, and sensor bias and precision compared to the laboratory analyzer (using Bland-Altman technique and linear regression analysis of pH, P_{aCO_2}, and P_{aO_2}). RESULTS: Duration of sensor use ranged from 1 to 304 hours (3–304 h in high position and 1–91 h in low position). Nine sensors were used for > 72 h (1 in low position and 8 in high position). Nine sensors were placed in low position, with a placement success rate of 56%. Eighteen sensors were placed in high position, with a placement success rate of 89%. The sensor values for pH, P_{aCO_2}, and P_{aO_2} were strongly correlated ($p < 0.0001$) with the laboratory-determined values. Bias and precision for all values met standards for discrete analyzers for laboratory use. CONCLUSIONS: The Neotrend device was accurate and reliable in the neonatal setting, allowing blood gas assessment with no iatrogenic blood loss. Catheter placement in high position may increase the likelihood of successful sensor placement and sensor duration of function. *Key words: intravascular blood gas sensor, umbilical artery catheter, monitoring, respiratory failure, neonates.* [Respir Care 2002;47(6):682–687]

Introduction

Arterial blood gas (ABG) values are a necessary diagnostic measurement in the management of critically ill, ventilated patients in the neonatal intensive care unit (NICU). The frequency of blood gas sampling is usually determined by the severity of the condition and clinical practice. A drawback to scheduled ABG sampling is that changing conditions may be missed. The relationship between P_{aCO_2} fluctuations and cerebral blood flow have been well described.[1,2] Hypercarbia and hypocarbia are both linked to the development of intraventricular hemorrhage and periventricular leukomalacia.[3–6] Frequent ABG sampling from a neonate results in blood loss that can necessitate transfusions and increase the risk of intravascular contamination. In critical situations the practitioner may have to wait for an ABG sample to be drawn and taken to the laboratory, which may or may not be in close proximity to the NICU. Rapid changes in blood gas values

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can occur following surfactant administration[7] and during high-frequency ventilation.[8] With the advent of continuous ABG monitoring, treatment modalities can be proactive rather than reactive.

Continuous ABG estimations have been available in a variety of noninvasive forms. Unfortunately, transcutaneous monitoring of carbon dioxide and oxygen levels necessitates frequent site changes and recalibration of the probe, and it is contraindicated in hemodynamically compromised patients because of changes in blood flow to the tissues.[9] Pulse oximetry offers continuous estimation of blood oxygen saturation but gives no information about pH or P_{aCO_2}. For the past 2 decades, continuous intra-arterial P_{aO_2} measurement has been available with the use of a Clark electrode with an umbilical artery catheter (UAC).[10] In recent years, advances in fiberoptics have provided continuous monitoring of blood gases in adults by allowing passage of a multivariable sensor through a catheter placed in a peripheral artery. Recently that same technology was adapted to allow sensor placement through the UAC in critically ill newborns.[11]

We evaluated a continuous-readout intravascular blood gas monitoring system (Neotrend, Diametrics Medical, St Paul, Minnesota) in critically ill newborns and compared the values obtained to ABG measurements made in the hospital's clinical laboratory. We also collected data to evaluate duration of sensor function and sensor performance in high UAC position (T8–10) versus low UAC position (L3–4). We hypothesized that the Neotrend intravascular blood gas monitoring system would produce blood gas results with clinically acceptable bias and precision, in comparison to laboratory values, but with no blood loss.

Methods

This study was reviewed and approved by the Children's Hospitals and Clinics institutional review board, and informed parental consent was obtained for each study participant. Patients who required UAC insertion were eligible for study entry. Patients suffering from known lethal anomalies, from any type of hypercoagulopathy, or in whom the need for arterial sampling was expected to be < 72 hours were excluded. Proper UAC placement was radiographically confirmed before sensor calibration.

The Neotrend sensor is a fiberoptic device that uses fluorescent technology to measure P_{aO_2}, optical absorption to measure pH and P_{aCO_2}, and a thermocouple to measure temperature (Fig. 1). Bicarbonate, base excess, and blood oxygen saturation are calculated. The sensor is 0.5 mm in diameter and is advanced 20 mm beyond the distal end of the UAC, residing in the descending aorta. Blood pressure monitoring, drug or flush infusion, and discrete blood sampling are possible with the sensor in situ. The sensor has a heparin coating that inhibits protein and thrombus formation.[12]

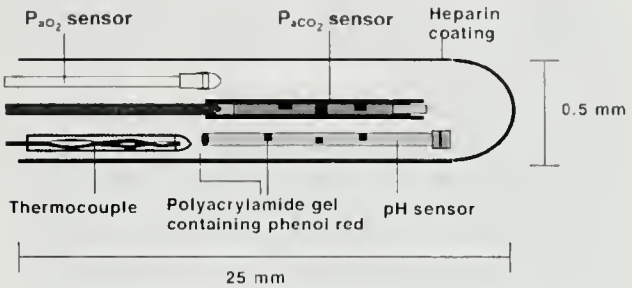

Fig. 1. Cross-section of the sensor tip of the Neotrend blood gas monitoring system. (Drawing courtesy of Diametrics Medical)

Sensor insertion was performed according to the manufacturer's recommendations. Before sensor insertion the device was calibrated with precision gases bubbled in sequence through the tonometer, under microprocessor control. Sensor calibration takes 31 minutes. Following calibration the sensor was flushed with normal saline, to ensure it remained in a liquid medium, and withdrawn from the tonometer, making sure the pressure port had no bubbles in it. The pressure port has a dead space of 0.1 mL. The UAC was clamped and the sensor was placed on the end of the UAC. A stopcock was attached to the pressure port and the pressure tubing was reconnected. The clamp was removed and the sensor was gently inserted through the UAC. Arterial line solutions used were normal saline (n = 18), 5% dextrose (n = 4), and 10% dextrose (n = 1), all with heparin concentrations of 0.25 units/mL. Following sensor insertion another radiograph was taken to recheck UAC position and sensor extension from the UAC. The sensor was allowed to equilibrate for no less than 20 min before the first comparison was made.

According to the manufacturer's guidelines, the first blood gas comparison following insertion was considered an in vivo correlation analysis, to identify any initial bias between the laboratory analyzer and the Neotrend sensor. This first comparison was eliminated from the statistical analysis, whether a bias adjustment was made or not. Criteria for adjustments were: pH difference > 0.03 units, P_{aCO_2} difference > 5 mm Hg, or P_{aO_2} difference > 10 mm Hg. After this initial bias adjustment, arterial blood samples were obtained as clinically indicated, and the values displayed on the Neotrend monitor were recorded. For all comparative ABG samples the patient was quiet and no ventilator changes were made in the 10 min prior to sampling. Monitors were set to read all blood gas values at 37° C. All blood gas samples were drawn by the attending nurse and were immediately brought to the laboratory for analysis by a laboratory technician. All samples were analyzed within 5 min. ABG samples were analyzed using a standard laboratory analyzer (ABL 725, Radiometer,

Copenhagen, Denmark). The respiratory therapist assigned to the NICU recorded the laboratory values and the Neotrend monitor data.

At the onset of the study it was our usual practice to place UACs in low position (L3–4). As the study progressed we placed some catheters in high position (T8–10). Though this study was not designed to compare values obtained from high and low sensor positions, we present and evaluate the data obtained from the high and low measurement sites.

We used Bland-Altman analysis to determine the degree of agreement between the 2 measurement methods.[13] Results were also analyzed using linear regression. Comparisons were made for pH, P_{aCO_2} and P_{aO_2}. The duration of proper sensor functioning and ease of placement were also documented. Successful and failed sensor insertions were recorded, as well as the duration (in hours) of sensor functioning. Sensors that fail before removal of the UAC can be replaced. All failed sensors were returned to the manufacturer for examination.

Results

Twenty-three neonates (57% male) were enrolled. Mean ± SD gestational age was 30.8 ± 6.3 weeks (range 23–40 wk). Mean ± SD birth weight was 1,759 ± 1,273 g (range 490–4,300 g). Twenty-seven sensor placement attempts were made, of which 21 (78%) were successful. Table 1 shows gestational age, birth weight, and catheter/sensor placement data.

The duration of sensor use ranged from 1 to 304 hours (from 3 to 304 hours in high position and from 1 to 91 hours in low position). Nine sensors were used for > 72 hours (1 in low and 8 in high position). One sensor failed 25 hours following insertion. Ten sensors functioned well until the infants no longer needed a UAC. Eleven sensors were removed prior to UAC discontinuation. Two UACs were inadvertently dislodged and the resulting UAC position was deemed inappropriate for sensor use. Blood could not be drawn from 3 UACs with the sensor extended. Two sensors were removed at the discretion of the attending physician. Three sensor cables were inadvertently disconnected. One sensor failed. Initial bias adjustments were made for 2 of 21 sensors.

A total of 244 blood samples were drawn for comparison with the sensor readings (219 with the UAC in high position and 25 with the UAC in low position). Figures 2–4 show Bland-Altman[13] plots for each variable, in high position, low position, and combined positions. For pH, bias was 0.03 in the low UAC position (see Fig. 2A) and zero in the high position (see Fig. 2B), which suggests better agreement between laboratory and Neotrend values measured in the high position ($p < 0.001$). The pH precision was ± 0.08 in low position and ± 0.06 at 2 standard deviations in the high position. Figure 2C shows bias of zero and precision of ± 0.06 at 2 standard deviations for combined high and low UAC placement for pH. Figure 3C shows bias of 3 and precision of 8 at 2 standard deviations for combined high and low P_{aCO_2}. Figure 4C shows bias of 4 and precision of ± 27 at 2 standard deviations for P_{aO_2} in combined high and low position.

Neotrend pH data were within ± 0.03 of the laboratory measurements. P_{aCO_2} values were within ± 4 mm Hg. Though intravascular monitoring devices are not required to meet standards for laboratory instruments, the pH and P_{aCO_2} data from the Neotrend sensor fall within the accuracy range required for discrete blood gas analyzers.

Discussion

In this study we evaluated an intravascular continuous blood gas monitor with a sensor designed for neonatal use, and compared it to a laboratory reference system. The system was easy to use and accurate. Though we did not randomize patients to high or low catheter position, our data suggest that the sensor is easier to insert and performs better when the UAC is placed in high position.

We had a wide range of blood gases for comparison. We studied patients with blood gas values throughout the range seen clinically, with pH values from 7.16 to 7.58, P_{aCO_2}

Table 1. Gestational Age, Birth Weight, and Sensor Position

	Low Position*	High Position†	All
Gestational age (mean ± SD wk)	34.9 ± 6	28.2 ± 5.1	30.8 ± 6.3
Gestational age range (wk)	24–40	23–40	23–40
Birth weight (mean ± SD g)	2,453 ± 1,304	1,314 ± 1,071	1,759 ± 1,273
Birth weight range (g)	520–4,300	490–3,844	490–4,300
Successful placement (*n*)	5 (56%)‡	16 (89%)‡	21 (78%)
Failed placement (*n*)	4 (44%)	2 (11%)	6 (22%)

* Low position of umbilical artery catheter was between L3 and L4
† High position of umbilical artery catheter was between T8 and T10
‡ High placement versus low placement $p = 0.1358$ using Fisher's exact test

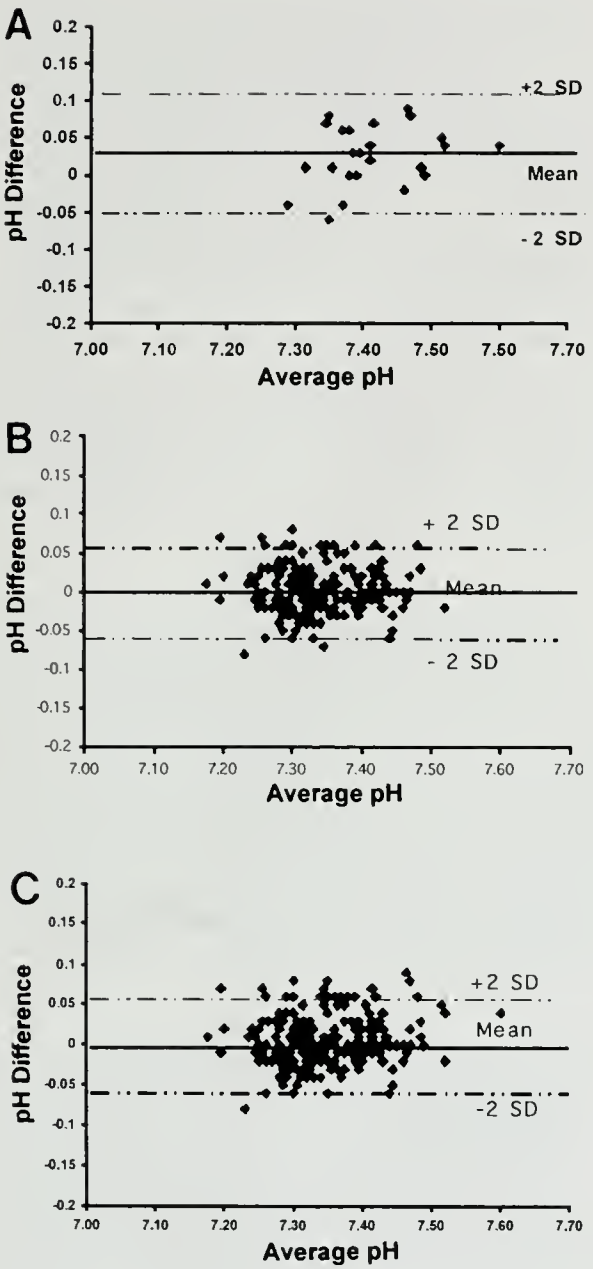

Fig. 2. Bias and precision of pH measurements with the Neotrend blood gas monitoring system. The pH difference is the laboratory value minus the Neotrend sensor value. The average pH is the average of the laboratory and Neotrend values. The bias is plotted as the solid horizontal line. Values closest to zero indicate minimal bias, or better agreement. The dotted lines represent ± 2 standard deviations, or the 95% confidence limits (precision). A. Values obtained with catheters in low position (L3–L4). B. Values obtained with catheters in high position (T8–T10). C. Combined values.

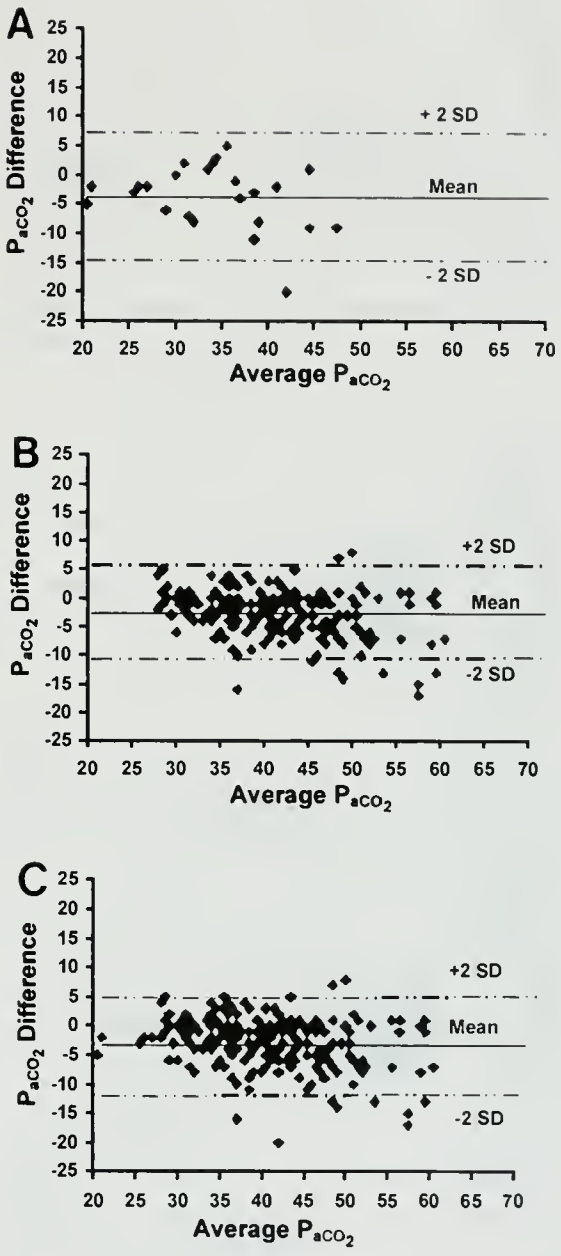

Fig. 3. Bias and precision of P_{aCO_2} measurements with the Neotrend blood gas monitoring system. The P_{aCO_2} difference is the laboratory value minus the Neotrend sensor value. The average P_{aCO_2} is the average of the laboratory and Neotrend values. The bias is plotted as the solid horizontal line. Values closest to zero indicate minimal bias, or better agreement. The dotted lines represent ± 2 standard deviations, or the 95% confidence limits (precision). A. Values obtained with catheters in low position (L3–L4). B. Values obtained with catheters in high position (T8–T10). C. Combined values.

values from 23 to 66 mm Hg, and P_{aO_2} values from 25 to 205 mm Hg. The system performed well over the entire clinical range.

Other fiberoptic blood gas monitors have been studied and found to produce accurate information.[14–16] The Neotrend system has a key difference from those previously reported: no blood is withdrawn from the patient's vascular system at any time during monitoring. Thus, true continuous monitoring can be achieved without any added

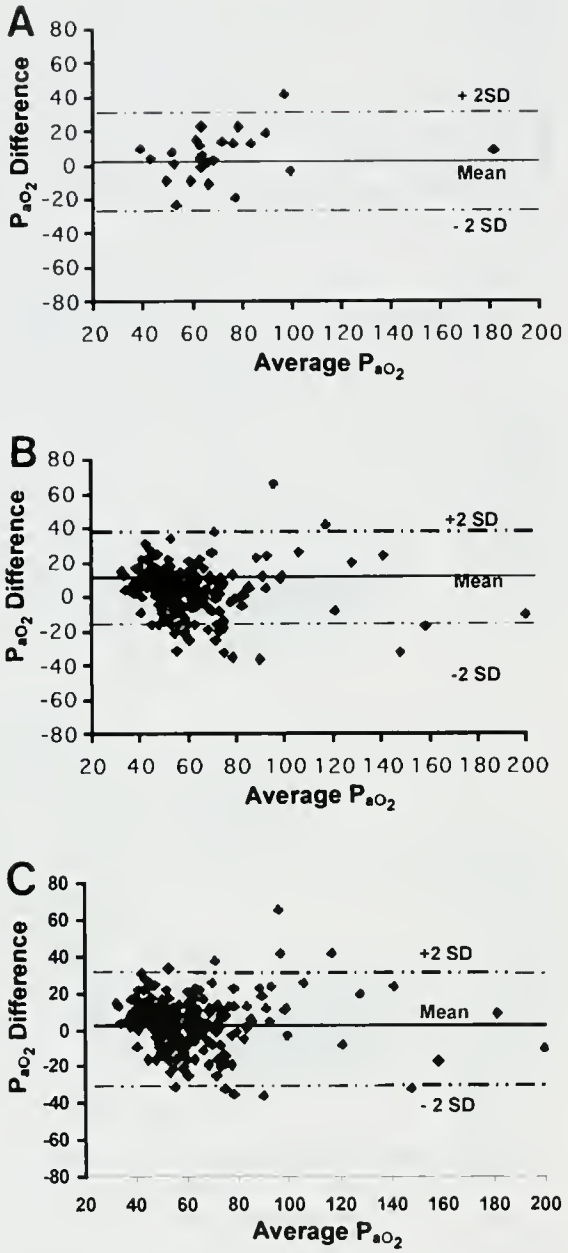

Fig. 4. Bias and precision of P_{aO_2} measurements with the Neotrend blood gas monitoring system. The P_{aO_2} difference is the laboratory value minus the Neotrend sensor value. The average P_{aO_2} is the average of the laboratory and Neotrend values. The bias is plotted as the solid horizontal line. Values closest to zero indicate minimal bias, or better agreement. The dotted lines represent ± 2 standard deviations, or the 95% confidence limits (precision). A. Values obtained with catheters in low position (L3–L4). B. Values obtained with catheters in high position (T8–T10). C. Combined values.

risk from blood loss or contamination other than that implicit with an indwelling catheter. In pre-term infants, even the brief hemodynamic change resulting from blood withdrawal into an analyzer and its subsequent return after analysis might have clinical impact.

Potential limitations to this study include the known differences in results between different blood gas analyzers, the relatively small number of patients studied, and the lack of randomization between high and low catheter position. Specimen transport to the laboratory can result in measurement inaccuracy, which increases with increasing time and distance.[16,17] Likewise, red cell sedimentation, excessive heparin, or air bubbles within the specimen are all potential confounders that do not occur with an indwelling continuous system. Our blood specimens were processed within 5 min of collection, but it is possible that different results might have been obtained if a bedside laboratory blood gas analyzer had been used. Our results suggest that catheter position impacts both ease of sensor placement and accuracy of results, but a prospective, randomized study would be necessary to test that hypothesis.

Conclusions

We found the Neotrend fiberoptic continuous blood gas monitoring system safe, easy to use, and accurate in neonates suffering respiratory failure. In addition to the valuable clinical information provided by a continuous monitoring system, there is great potential for the safer use of mechanical ventilation, decreased iatrogenic blood loss and donor exposure, and cost savings. Continuous ABG monitoring will not completely replace laboratory sampling, but, like continuous measurement of oxygen saturation at the bedside, it may extend the clinician's ability to recognize and intervene in complex clinical problems.

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Independent Lung Ventilation in a Patient with Complex Congenital Heart Disease

Donna Levine MEd RRT, Tina Carmichael RRT, and Peter C Laussen MBBS

Independent lung ventilation (ILV) is recognized as a method of treating unilateral lung disease. We report the use of ILV in a 22-year-old woman with acute respiratory failure and complex congenital heart disease with different sources of left and right pulmonary blood flow. She had a palliated single-ventricle circulation with pulsatile pulmonary blood flow from an aorto-pulmonary central shunt to her left lung and nonpulsatile pulmonary blood flow via a classic Glenn shunt (superior vena cava to right pulmonary artery). On admission she was hypoxemic and hypotensive. Her chest radiograph revealed opacification of the left lung and hyperinflation of the right lung, which was more compliant than the left lung. Following placement of a double-lumen endotracheal tube, synchronized ILV was instituted. ILV allowed us to deliver lower ventilator pressure to the right lung, which alleviated the over-distention of the right lung (to which pulmonary blood flow was supplied by the nonpulsatile Glenn shunt) while higher airway pressures were delivered to the diseased left lung, to facilitate re-expansion. There was immediate improvement in gas exchange and blood pressure. After 3 days the double-lumen endotracheal tube was changed to a single-lumen tube. She was extubated on day 6 and discharged on day 13. This case demonstrates the advantage of ILV in a patient with abnormal pulmonary blood flow and different lung mechanics in the left and right lungs. *Key words: independent lung ventilation, ILV, congenital heart disease, Waterston shunt, Glenn shunt, double-lumen endotracheal tube.* [Respir Care 2002;47(6):688–692]

Introduction

Independent lung ventilation (ILV) is defined as ventilating the left and right lungs selectively. ILV is beneficial in treating unilateral lung disease, when there are substantial differences in lung compliance or pulmonary blood flow to the right and left lungs.[1,2] The first reported use of ILV was in 1931, for thoracic surgery.[2] Although not widely used in the critical care setting, ILV has proven successful for patients with asymmetric lung disease, particularly in the setting of refractory hypoxemia or a major deterioration in cardiac output from increased intrathoracic pressure secondary to positive-pressure ventilation (PPV) applied to both lungs. This may be especially important for patients with different sources of left and right pulmonary blood flow.

Although outcomes for patients with all types of congenital heart disease have substantially improved over the years, previous palliative surgery or sequelae following corrective surgery may be associated with important physiologic limitations, particularly with regard to pulmonary blood flow. This case report describes an adult with palliated congenital heart disease and different sources of pulmonary blood flow, and in whom ILV enabled successful treatment of acute respiratory failure.

Case Summary

A 22-year-old, 53-kg woman with complex palliated congenital heart disease was referred to our institution for

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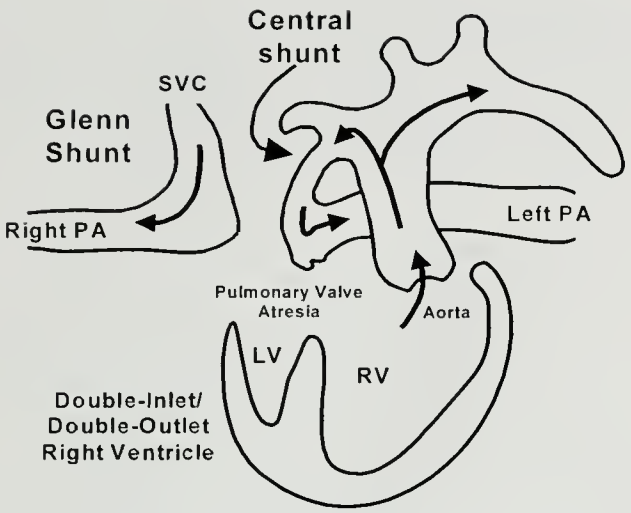

Fig. 1. Basic cardiac anatomy and origin of right and left pulmonary blood supply. This patient had a double-inlet/double-outlet right ventricle with pulmonary atresia, blood flow to the right lung through a Glenn shunt (superior vena cava anastomosis to the right pulmonary artery), and blood flow to the left lung through a central shunt (synthetic tube graft from the innominate artery to the left pulmonary artery). PA = pulmonary artery. SVC = superior vena cava. RV = right ventricle. LV = left ventricle.

consideration of extracorporeal membrane oxygenation because of left lung consolidation and acute respiratory failure. She presented initially to her local emergency room with complaints of a nonproductive cough associated with fever, myalgia, and arthralgia for 2 days. On examination she was deeply cyanotic, tachypneic, and had chest wall retractions from increased work of breathing. Blood oxygen saturation measured via pulse oximetry was 52% while patient breathed supplemental oxygen via nasal cannula. An admission chest radiograph showed complete opacification of the left lung, and a subsequent computed tomogram showed consolidation of the left lung, without pleural fluid. Because of acute respiratory distress and severe hypoxemia she was intubated, following which she became hypotensive and required support with fluid replacement and increasing vasoactive support.

Her cardiac diagnosis included a double-inlet/double-outlet right ventricle, hypoplastic left ventricle, multiple ventricular septal defects, and pulmonary valve atresia. Essentially she had a single ventricle (morphologic right ventricle) (Fig. 1). Because of pulmonary atresia and hypoplasia of the pulmonary ventricle, as a neonate she had pulmonary blood flow established by placement of a Waterston shunt from the ascending aorta to the right pulmonary artery. At age 7 the blood supply to the right and left pulmonary arteries were separated, constructing a classic Glenn shunt (superior vena cava to right pulmonary artery, disconnecting the right and left pulmonary arteries) and maintaining blood supply to the left pulmonary artery via a central shunt created from the ascending aorta to the left pulmonary artery. Because of the high pressures to the left pulmonary artery from the central shunt, she developed pulmonary vascular obstructive disease to the left pulmonary circulation and was therefore deemed unsuitable for a modified Fontan procedure. However, serial echocardiograms continued to show good right systemic ventricle systolic function, without substantial atrioventricular or semilunar valvular insufficiency. Her baseline oxygen saturation was 85%. She led an active lifestyle and attended college. The Glenn shunt supplied blood flow to the right middle and lower lobes. The aortic-to-pulmonary artery central shunt supplied pulsatile blood flow to the left lung and the right upper lobe (see Fig. 1). Pulmonary blood flow to regions supplied by the Glenn shunt was therefore nonpulsatile and dependent on the transpulmonary pressure gradient from the superior vena cava to the atrium.

After transfer from an out-of-state community hospital and on arrival to our intensive care unit, the patient was deeply cyanotic, with poor extremity perfusion and diminished aeration of the left lung by auscultation. She was receiving dopamine at 20 μg/kg/min and epinephrine 1

Fig. 2. Chest radiograph at admission. A single-lumen endotracheal tube is in place. Note opacification of the left lung and hyperinflation of the right lung. At this point, ventilator peak inspiratory pressure was 31 cm H_2O, PEEP was 10 cm H_2O, mean airway pressure was 16 cm H_2O, fraction of inspired oxygen was 1.0, and blood oxygen saturation (measured via pulse oximetry) was 55%.

Table 1. Ventilation Settings and Gas Exchange Before, During, and After Independent Lung Ventilation

	PIP/PEEP (cm H_2O)	$\bar{P}_{aw}$ (cm H_2O)	V_T (cc)	f (breaths/min)	F_{IO_2}	S_{pO_2} (%)	pH	P_{aCO_2} (mm Hg)	P_{aO_2} (mm Hg)
Before ILV (single-lumen ET)	31/10	16.1	500	30	1.00	55	7.39	45	32
At 12 hours of ILV	L 32/10	17.3	250	25	0.80	71	7.51	27	41
	R 16/4	7.7	265	25	0.80				
At 24 hours of ILV	L 30/10	12.0	220	12	0.55	80	7.40	49	49
	R 16/4	6.2	380	12	0.55				
At 72 hours of ILV	L 17/4	5.3	220	12	0.40	88	7.42	48	55
	R 15/4	5.2	280	12	0.40				
Single-lumen ETT	25/4	7.0	500	12	0.50	83	7.41	47	51
After extubation					0.45	89	7.48	33	46

PIP = peak inspiratory pressure
PEEP = positive end-expiratory pressure
$\bar{P}_{aw}$ = mean airway pressure
V_T = tidal volume
f = respiratory rate
F_{IO_2} = fraction of inspired oxygen
S_{pO_2} = blood oxygen saturation measured via pulse oximetry
ILV = independent lung ventilation
ET = endotracheal tube
PBF = pulmonary blood flow

μg/kg/min to maintain a systolic blood pressure of 90–100 mm Hg. She was intubated and ventilated by hand, with a high peak inspiratory pressure and initial blood gas values (while on 100% oxygen) of pH 7.15, P_{aCO_2} 44 mm Hg, and P_{aO_2} 42 mm Hg. Because of her underlying cardiac pathology, the high peak inspiratory and resultant mean intrathoracic pressure compromised her cardiac output by severely reducing venous return and blood flow through the Glenn shunt to the systemic ventricle. She had pulmonary blood flow to her left lung via the central shunt, although the chest radiograph showed complete opacification because of collapse and consolidation. The right lung was preferably ventilated because of its higher compliance, and the chest radiograph showed that it was hyperinflated (Fig. 2). She therefore had ventilation but limited perfusion to the right lung, and perfusion but limited ventilation to the left lung. She remained severely hypoxemic despite a high level of PPV (pressure-controlled mode with 32 cm H_2O inspiratory pressure and 10 cm H_2O positive end-expiratory pressure [PEEP]) and continued to require substantial inotrope and vasopressor support because of hypotension.

Because of the unilateral nature of her lung disease and the potential benefit of ventilating the right lung at lower airway pressures to optimize pulmonary blood flow through the Glenn shunt, a left-sided 35 French double-lumen endotracheal tube (ETT) was placed to enable ILV. Tube placement was confirmed by fiberoptic bronchoscopy.

Thick yellow secretions were aspirated during the bronchoscopy and analyzed. Broad-spectrum antibiotics were started. There were no complications associated with the double-lumen ETT intubation. Technical aspects of double-lumen ETTs have been reviewed by Berumol[3] and by Barash et al.[4]

To initiate mechanical ventilation the 2 lumens were connected to separate Servo 900C ventilators (Siemens, Solna, Sweden); a "master-slave cable" connector between

Fig. 3. Chest radiograph 1 day after admission. A left-sided double-lumen endotracheal tube is in place for independent lung ventilation. At this point the left lung mean airway pressure was 12 cm H_2O, and the left lung has re-expanded. In the right lung mean airway pressure was 6 cm H_2O, and the right lung is less hyperinflated. At this point, fraction of inspired oxygen was 0.55 and blood oxygen saturation (measured via pulse oximetry) was 80%.

the 2 ventilators allowed simultaneous initiation of inspiration to both lungs. The left lung was ventilated using a time-cycled, pressure-controlled mode and PEEP, to generate an expiratory tidal volume of 5 mL/kg. The right lung was also ventilated with that mode, but with a lower airway pressure and PEEP, to maintain the same tidal volume of 5 mL/kg.

Table 1 shows the airway pressures and gas exchange values before, during, and after ILV. Within 24 hours oxygenation had substantially improved. Selectively ventilating the left lung at a high mean airway pressure re-expanded the left lung, with chest radiograph showing near complete resolution of the consolidation process (Fig. 3). Ventilating the right lung at a lower mean intrathoracic pressure improved pulmonary blood flow through the Glenn shunt and therefore preload to the systemic ventricle. Systemic blood pressure and perfusion improved, and the dopamine and epinephrine infusions were rapidly weaned once ILV was established. As the left lung pathology resolved, conventional mechanical ventilatory support through a single-lumen ETT was resumed after 72 hours, and the patient was successfully extubated after 150 hours. The patient was discharged 13 days after admission, with blood oxygen saturation (on room air) back to baseline (86%). She graduated from college 6 months following discharge from the hospital.

Discussion

This case report highlights the difficulties that can occur during PPV in patients with complex congenital heart disease who have different sources of left and right pulmonary blood flow. Specifically, in our patient pulmonary blood flow to the left lung and right upper lobe was pulsatile from a central shunt connected to the aorta. Blood flow to the right middle and lower lung was nonpulsatile and dependent on venous pressure within the Glenn shunt.

Glenn was the first to show that systemic venous return could flow passively through the pulmonary circulation without requiring assistance from the right ventricle, when he constructed a unidirectional shunt as a palliative procedure for tricuspid atresia.[5]

The Glenn shunt is rarely performed today, as the pulmonary arteries are not in continuity and palliation is short-lived. In contrast, a complete cavopulmonary connection (Fontan procedure), is a commonly used repair for many forms of single-ventricle congenital heart disease. As with the Glenn shunt, the complete cavopulmonary connection enables effective pulmonary blood flow without requiring assistance from the right ventricle. Changes in mean intrathoracic pressure and pulmonary vascular resistance may therefore have an important effect on pulmonary blood flow in this circumstance.

Under ideal conditions a transpulmonary gradient of 5–10 mm Hg will allow adequate blood flow across the pulmonary vascular bed and sufficient preload to the systemic ventricle to maintain cardiac output. Pulmonary blood flow is biphasic following a cavopulmonary connection, and early resumption of spontaneous ventilation is recommended to offset the detrimental effects of PPV.[6] Doppler analysis has shown that pulmonary blood flow occurs predominantly during inspiration in a spontaneously breathing patient (ie, when the mean intrathoracic pressure is subatmospheric).[6,7] The use of PEEP is also debated in these patients. The beneficial effects of increasing functional residual capacity, maintaining lung volume, and redistributing lung water need to be balanced against the possible detrimental effects of increasing mean intrathoracic pressure.

In our patient, who had regional differences in pulmonary blood flow, ILV improved gas exchange and systemic hemodynamics in the setting of asymmetric lung disease. The higher lung compliance and lower airways resistance in the unaffected right lung meant that during conventional mechanical ventilation with a time-cycled, pressure-controlled mode most of the ventilation was distributed to the right lung, resulting in hyperinflation and elevated mean intrathoracic pressure, which adversely affected pulmonary blood flow. In addition, preload to the systemic ventricle was diminished, which contributed to low cardiac output and hypotension.

ILV provided appropriate tidal volume delivery to the right lung at much lower mean intrathoracic pressure, which immediately improved pulmonary blood flow through the Glenn shunt and improved gas exchange and preload to the single ventricle. As a result, inotrope and vasopressor support was weaned soon after ILV was established. A much higher mean intrathoracic pressure was delivered to the diseased left lung, without compromising gas exchange or cardiac output, which enabled re-expansion of the left lung and rapid resolution of the underlying process.

This case demonstrates the advantage of ILV in a patient with different sources of left and right pulmonary blood flow and altered lung mechanics. Despite the complex nature of severe congenital cardiac defects and their palliations, many patients are able to maintain relatively normal activity and lifestyle. However, during acute illness, important disturbances can occur, contributing to acute cardiorespiratory failure, as demonstrated by our patient. The complex physiology of many palliated congenital cardiac defects means that close collaboration between the intensivist, cardiologist, and respiratory therapist is mandatory when planning management strategies for these patients.

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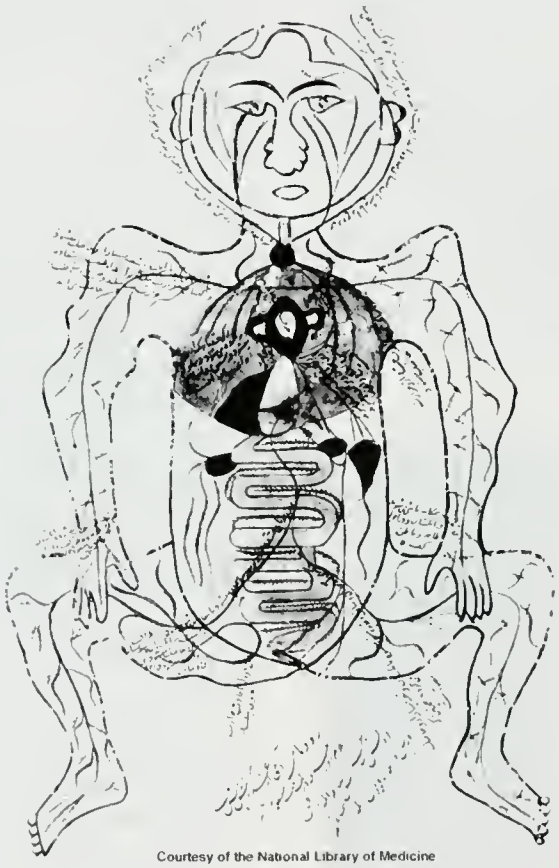

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Unilateral Expiratory Airflow Obstruction During Forced Exhalation

Umur Hatipoglu MD, Franco Laghi MD, Steven E Cattapan MD, and Arcot J Chandrasekhar MD

We present a case of unilateral airflow obstruction during forced exhalations. The patient presented with episodic dyspnea and wheezing, particularly when he lay in the right lateral decubitus position. Spirometry revealed symmetric, marked reductions in forced expiratory volume in the first second and forced vital capacity values, while plethysmography demonstrated a near-normal total lung capacity. Bronchoscopy revealed a polypoid lesion in the right main bronchus, of which biopsy specimens demonstrated adenoid cystic carcinoma. Following a right pneumonectomy, total lung capacity was markedly reduced; interestingly, however, spirometry was essentially unchanged. This case is an elegant illustration of the effects of respiratory maneuvers and body position on airway caliber. *Key words: adenoid cystic carcinoma, pulmonary function testing, PFT, airflow obstruction.* [Respir Care 2002;47(6):693–695]

Introduction

This case describes a 60-year-old nonsmoking man with cystic adenoid carcinoma obstructing the right main bronchus. His presenting symptoms misled his physicians to believe he had asthma. One prominent finding was that the patient had more pronounced symptoms when in right lateral decubitus position—a result of positional changes in airway caliber. Pulmonary function testing was also consistent with obliteration of airflow through the right main bronchus when airway caliber was reduced, such as during the forced vital capacity maneuver or when expiring below the functional residual capacity. Bronchoscopy provided the correct diagnosis.

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Case Report

A 60-year-old man presented with episodic, progressive shortness of breath and infrequent hemoptysis over a 3-year period. The patient did not complain of orthopnea, but he did feel more dyspneic when lying on his right side. Occasional wheezing led to a diagnosis of asthma, with unsuccessful treatment. He was otherwise healthy, used no medications, and had no allergies. His medical history was notable for borderline hypertension. He had never smoked and did not consume alcohol. On physical examination the patient was a thin man in no distress, with unlabored breathing. His blood pressure was 130/80 mm Hg, his pulse was 76 beats/min, and his respiratory rate was 18 breaths/min. Chest auscultation revealed wheezing, but the remainder of his examination was unremarkable.

Table 1 shows the results of the pulmonary function tests. The first chest radiograph showed slight elevation of the right hemidiaphragm (Fig. 1).

A bronchoscopy was performed to investigate the etiology of the hemoptysis, and a large tan-pink, lobulated, polypoid, soft lesion was found in the right main bronchus. The bronchoscope could not be advanced beyond the lesion. The lesion moved during respiration, with complete occlusion of the airway during forceful exhalation (Fig. 2). Biopsy of the lesion revealed adenoid cystic carcinoma, cribriform type, for which the patient underwent a right pneumonectomy (Fig. 3). Because the operative specimen showed positive bronchial margins, the patient received adjuvant, computed tomography-guided, 3-dimensional, conformal radiotherapy to the right bronchial stump. Ninc

Table 1. Pulmonary Function Test Results

	Preoperative	Postoperative
FVC (L)	2.02 (53)*	1.75 (46)
FEV_1 (L)	1.6 (52)	1.46 (48)
FEV_1/FVC (%)	79 (99)	83 (105)
FRC (L)	3.16 (96)	1.71 (51)
ERV (L)	0.05 (4)	0.37 (31)
RV (L)	3.11 (149)	1.34 (63)
TLC (L)	5.28 (89)	3.09 (52)
RV/TLC (%)	58 (178)	43 (131)
$D_{LCO_{corr}}$ (mL/min/mm Hg)	18.5 (67)	15.18 (56)
D_L/V_A (ml/min/mm Hg/L)	5.9 (119)	6.27 (127)

*Values in parentheses are percent of predicted
FVC = forced vital capacity
FEV_1 = forced expiratory volume in one second
FRC = functional residual capacity
ERV = expiratory reserve volume
RV = residual volume.
TLC = total lung capacity.
$D_{LCO_{corr}}$ = diffusing capacity of the lung for carbon monoxide, corrected for hemoglobin.
D_L/V_A = diffusion per unit of alveolar volume.

Fig. 2. Right main bronchus during exhalation (left) and during inhalation (right). During exhalation the lobulated endobronchial adenoid cystic carcinoma occupies the entirety of the right main bronchus lumen. During inhalation there is a small, sickle-shaped opening in the ventral portion of the bronchus (near 12 o'clock position). Some pooling of blood can be seen in the dorsal portion of both photographs (about 4 o'clock position).

months after surgery, repeat pulmonary function testing showed similar values of forced expiratory volume in the first second and forced vital capacity (see Table 1). One and a half years after surgery, the patient felt well.

Discussion

A large mass that completely occludes an airway is associated with atelectasis distal to the lesion. Airway caliber, however, is also a function of respiratory dynamics. Exhalation is associated with a decrease in airway caliber; forceful exhalation with an even greater decrease. A lesion that permits inhalation but then occludes the airway during exhalation (when the airway caliber is less) creates a ball-valve phenomenon leading to hyperinflation. In our patient, however, the passage of air into and out of the right lung during tidal breathing was supported by a near-normal total lung capacity (as measured by plethysmography) and only minimal right lung atelectasis on the chest radiograph. As the patient tried to breathe below functional residual capacity, continued airway narrowing prevented further expiration from the right lung and led to low expiratory reserve volume and high residual volume.

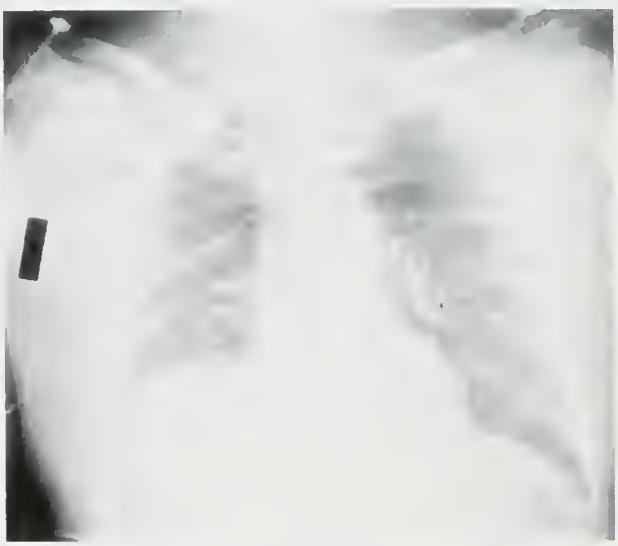

Fig. 1. Chest radiograph obtained before right pneumonectomy. Note that the right hemidiaphragm is elevated but the left lung field and mediastinum are normal.

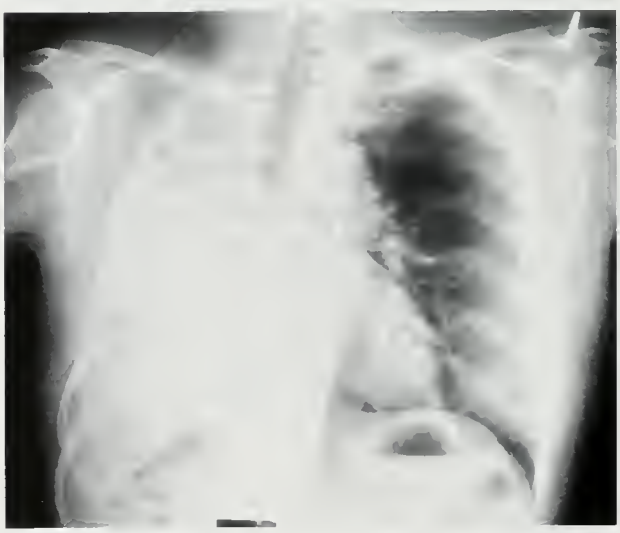

Fig. 3. Chest radiograph obtained after right pneumonectomy.

Forceful exhalation led to early and complete airway occlusion, as evidenced by the spirometric data (see Table 1). Preoperative forced expiratory volume in the first second and forced vital capacity were half of the predicted values; postoperative values were essentially unchanged. We conclude that because of early and complete airflow obstruction of the right lung during forceful exhalation, preoperative spirometry reflected the flow characteristics of only the left lung.

The plethysmographically-measured lung volume, in contrast to the lung volume calculated from spirometry results, was dramatically different following pneumonectomy (see Table 1). The disparate findings of spirometry and plethysmography are a result of the different methods of measurement. Spirometry measures the volume of air exhaled from the lungs, whereas plethysmography correlates changes in the pressure of air outside the subject's body to the subject's lung volumes; thus, any compressible gas—such as the air in this patient's right lung—is measured.

Changes in airway caliber were also probably responsible for the worse dyspnea in right lateral decubitus position. The gravitational effect of the mediastinum and abdominal contents decreases the lung volume of the dependent lung. Decreased lung volume is associated with decreased airway caliber and, in this patient, led to early occlusion of the right main bronchus. Moreover, perfusion increases to the dependent lung, thereby worsening ventilation-perfusion matching when he lay on his right side.

Primary adenoid cystic carcinoma of the lung typically presents with symptoms of bronchial obstruction and/or bronchial irritation, such as dyspnea, wheezing, cough, and increased sputum production. That constellation of symptoms may masquerade as asthma.[1,2] Tracheal location may also lead to stridor and the sensation of choking. Hemoptysis is also commonly present. Because of its insidious growth and clinical course, diagnosis is typically made late (ie, average symptom duration of 4.5 years).[3]

Primary adenoid cystic carcinoma of the lung has a relatively indolent course in most but not all cases. Survival with disease may extend for 18 years.[3] Treatment with complete resection of tumor leads to a relatively good prognosis; unfortunately, as these tumors tend to invade submucosally, positive surgical margins are common.[4] Following incomplete resection, adjuvant radiotherapy is often recommended.[5] Chemotherapy is reserved for metastatic disease.[6]

In summary, we present a case of unilateral complete airflow obstruction during forced exhalation. The lesion was not large enough to occlude the airway during resting tidal breathing, but it was large enough to completely occlude the airway during forceful exhalation. The effects of respiratory maneuvers and body position on airway caliber are elegantly demonstrated in this case. The cause of the obstruction was a primary adenoid cystic carcinoma of the lung, an extremely rare and often indolent cancer that can mimic asthma.

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Letters

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Is the Gastrointestinal Tract the Sole Source of Organisms in Ventilator-Associated Pneumonia?

Dr Charles Durbin's presentation, entitled "Evidence-Based Airway Management,"[1] to the RESPIRATORY CARE Journal Conference on Evidence-Based Medicine, and the discussion that ensued after the presentation (December 2001 issue of RESPIRATORY CARE) contain a wealth of information. Dr Durbin's numerous references incorporate ample evidence justifying the recommendations that he makes. In the discussion after Dr Durbin's presentation, Dean Hess PhD RRT FAARC, who served as Co-Chair of the conference, offered comments that challenge some of Dr Durbin's advisories. The RESPIRATORY CARE readership would be better served if we were provided with specific studies validating the points that Dr Hess listed. For example, the findings of the meta-analysis by Cook et al[2] are questioned, but Dr Hess doesn't provide any basis for his skepticism with regard to the Cook et al data. Dr Hess vocalizes his belief that the organisms that access the lower respiratory tract from outside the endotracheal/tracheostomy tube, rather than through the lumen thereof, are solely responsible for ventilator-associated pneumonia (VAP), but cites no authoritative references for such a claim. Many clinicians, in addition to Dr Durbin, have conceded that some VAP episodes are probably attributable to gastrointestinal flora, but few would contend that the gastrointestinal tract is the sole source of those pathogens.

Dr Hess goes on to voice his belief that the incidence of VAP is unrelated to the choice of humidification device (active or passive), circuit (heated or unheated), or type of humidification device (heated water-bath, heat and moisture exchanger, or heat and moisture exchanging filter) that is employed, citing "some newer evidence." Regrettably, we are not provided with any references that incorporate that newer evidence. Yet the single most authoritative study published to date that examines this issue, the randomized controlled trial by Kirton et al[3] (cited by Dr Durbin as his reference 41) is ignored. In face-to-face discussions of this topic that Dean Hess and I have undertaken in the past, he has correctly pointed out that the Kirton et al study is yet to be duplicated. Nevertheless, the methods used in that investigation were flawless and the results were published in a journal that enjoys an unexcelled reputation for being authoritative and comprehensive.

I have long been concerned about both patients' and caregivers' risk of exposure to bacterial microaerosols expelled from mechanical ventilators, and have elaborated on this worrisome issue in a recent editorial.[4] The placement of a competent heat and moisture exchanging filter in the ventilator circuit serves to protect both patients and caregivers from this hazard. I would respectfully suggest that water-bath humidifiers can facilitate the production of particulate aerosols laced with bacteria and viruses. However, Dr Hess has voiced his belief to me, in personal conversations, that the high temperatures achieved in the canisters of heated water-bath humidifiers can be expected to kill any organisms that might be mobilized into the inspiratory circuit. Yet certain clinical studies[5,6] contradict such a claim. Dean Hess has a well-deserved reputation for being a thoughtful clinician and a gifted writer. We would all benefit from any additional insights that he could furnish to us with respect to his clinical impressions.

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Associate Editor Dean R Hess responds:

And Now For the Rest of the Story

A concern related to the care of the mechanically ventilated patient is the development of VAP.[1–4] For many years, this concern focused on the ventilator circuit and humidifier. I can recall early in my career that we changed the circuit and humidifier daily, or even more frequently, in our attempt to decrease the rate of VAP. Little did we know at the time that such efforts probably contributed to an increased VAP rate. Mr Demers clearly has the belief that the patient and caregiver are best served by placing a filter and passive humidifier directly on the airway of the intubated mechanically ventilated patient. Because I review the scientific content of *CHEST* journal on a monthly basis, I was surprised that I missed Mr Demers' recent publication.[5] After I found it I noted that it is published under the heading "Opinions/Hypotheses." As I read the paper I must admit that my first response was that it is full of opinion but empty of science. In fact, I found the paper to be, more-or-less, an "infomercial" for the device that Mr Demers favors for use as a heat-and-moisture-exchanging filter. In this era of evidence-based medicine, we should not confuse speculation with science, and we must recognize that outcome data trumps expert opinion. In the spirit of evidence-based medicine, I will defend my position on this subject.

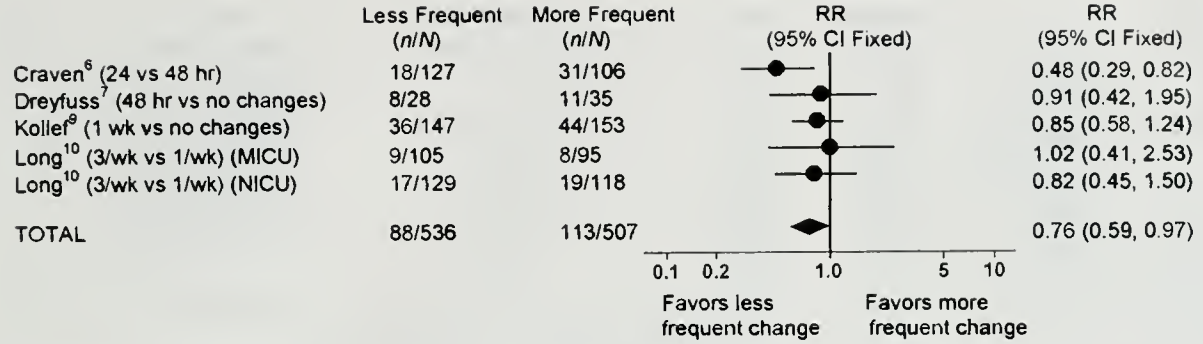
Less Frequent (n/N)
More Frequent (n/N)
RR (95% CI Fixed)
RR (95% CI Fixed)
Craven[6] (24 vs 48 hr) 18/127 31/106 0.48 (0.29, 0.82)
Dreyfuss[7] (48 hr vs no changes) 8/28 11/35 0.91 (0.42, 1.95)
Kollef[9] (1 wk vs no changes) 36/147 44/153 0.85 (0.58, 1.24)
Long[10] (3/wk vs 1/wk) (MICU) 9/105 8/95 1.02 (0.41, 2.53)
Long[10] (3/wk vs 1/wk) (NICU) 17/129 19/118 0.82 (0.45, 1.50)
TOTAL 88/536 113/507 0.76 (0.59, 0.97)
0.1 0.2 1.0 5 10
Favors less frequent change
Favors more frequent change

Fig. 1. A meta-analysis combining the results of randomized studies that compared ventilator-associated pneumonia (VAP) rate with extended ventilator circuit change intervals. RR = risk ratio. *n* = number of cases of VAP. *N* = total number of patients. CI = confidence interval. MICU = medical intensive care unit. NICU = neurologic intensive care unit.

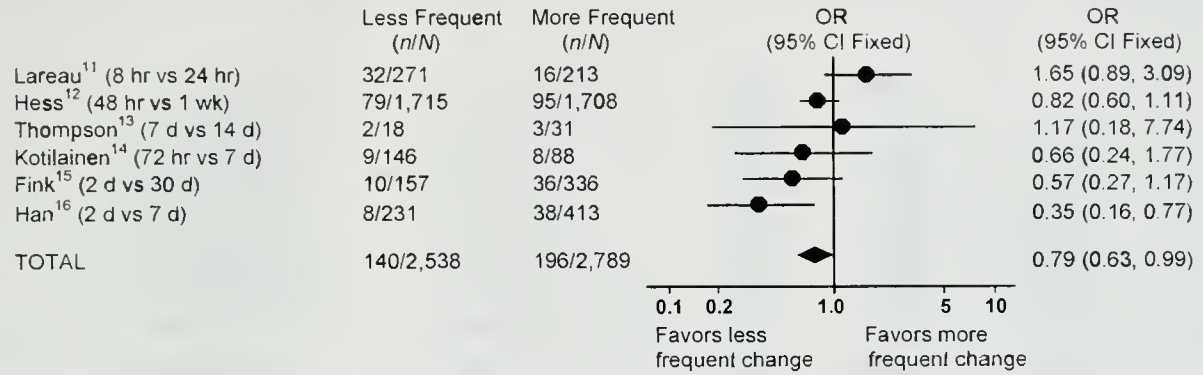

Fig. 2. A meta-analysis combining the results of observational studies that compared ventilator-associated pneumonia (VAP) rate with extended ventilator circuit change intervals. OR = odds ratio. *n* = number of cases of VAP. *N* = total number of patients. CI = confidence interval.

VAP may arise from either what is breathed through the endotracheal tube (ETT) or what is aspirated around the cuff of the ETT. The available evidence suggests that aspiration of gastric, oropharyngeal, and sinus secretions are more important in the pathogenesis of VAP than what is breathed through the ETT.[1,2] Ventilator circuits connected to the ETT are contaminated. However, the organisms cultured from the ventilator circuit are almost always from the patient.[6,7] Dreyfuss et al[7] reported that colonization of the Cascade humidifier was a rare event. Moreover, colonization of the pharynx preceded colonization of the ventilator circuit in nearly every case. To quote the exact words of Dreyfuss, "the etiologic agents of pneumonia were recovered from the circuits always after being detected first in patients."[7] In other words, the patient contaminates the ventilator circuit rather than vice versa. Is the purpose of a filter between the patient and the ventilator circuit to protect the ventilator circuit from the patient? If so, the ventilator circuit thanks you for your efforts. However, I have yet to witness the death of a ventilator circuit due to this.

Although it is possible that the source of VAP may be what is breathed from the ventilator circuit through the ETT, there are at least 3 arguments to support that VAP is more likely due to what is aspirated around the cuff of the ETT.

1. Ten studies[7,16] report no significant increase in VAP with less frequent ventilator circuit changes. In fact, a meta-analysis of those studies shows that the combined VAP rate is lower with less frequent circuit changes (Figs. 1 and 2). This contradicts the assertion that the ventilator circuit and humidifier contribute a significant risk for the development of VAP.

2. The stomach is a source of bacteria that produce VAP.[1,2] Stress ulcer prophylaxis that maintains gastric sterility decreases the VAP rate.[17,19] Randomized trials report that the rate of aspiration around the cuff of the ETT is decreased when patients are placed into a semi-recumbent position rather than a supine position.[20,21]

3. Randomized trials report a significant decrease in VAP with subglottic secretion clearance, presumably because of reduced aspiration around the cuff.[22–25]

Mr Demers' implication of microaerosol-generating cascade humidifiers in the delivery of contaminants into the lower respiratory tract is antiquated, because those devices are no longer manufactured. Most of us are using heated passover or wick humidifiers in our ventilator circuits.

I did a meta-analysis of randomized studies comparing VAP rate with passive versus active humidification (Fig. 3).[26–32] Indeed, consistent with Mr Demers's bias, there is a combined effect of a significant reduction in VAP with the use of a passive humidifier—although much of this effect is due to the trial by Kirton et al[31] that Mr Demers so fondly uses in support of his position. However, there are other impor-

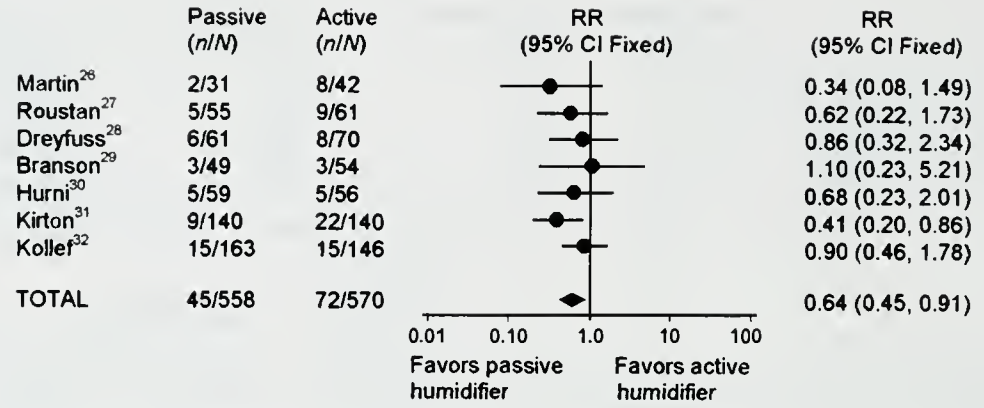

Fig. 3. A meta-analysis combining the results of randomized studies that compared ventilator-associated pneumonia (VAP) rate with passive and active humidification during mechanical ventilation. RR = risk ratio. *n* = number of cases of VAP. *N* = total number of patients. CI = confidence interval.

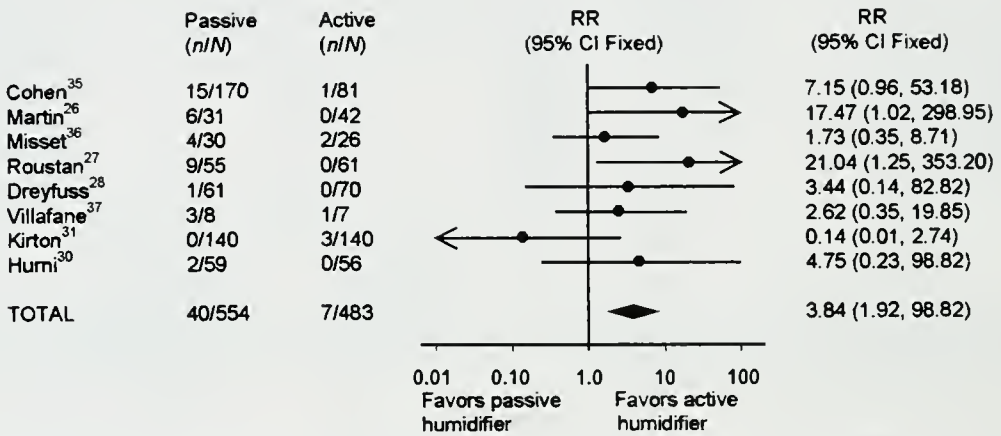

Fig. 4. A meta-analysis combining the results of randomized studies that compared the rate of endotracheal tube occlusion with passive and active humidification during mechanical ventilation. Note a significantly higher rate of airway occlusion with the passive humidifier. In fact, the rate of airway occlusion is higher with passive humidification in every study but one. RR = risk ratio. *n* = number of cases of VAP. *N* = total number of patients. CI = confidence interval.

tant issues that must be considered when a passive humidifier is used. These include the dead space of the device, the resistive load of the device, the difficulty in delivery of aerosolized medications, and the potential for airway occlusion. The mechanical dead space of the passive humidifier can be substantial—particularly when using tidal volume of 6 mL/kg predicted body weight as a lung-protective strategy. The dead space of the passive humidifier may amount to 25% or more of the delivered tidal volume. Pelosi et al[33] reported increased work of breathing attributable to the use of a passive humidifier. Le Bourdelles et al[34] reported a higher P_{aCO_2} and higher minute ventilation requirement with the use of a passive humidifier. To deliver aerosolized medications the filter and humidifier must be removed from the circuit—this negates all of the caregiver benefits espoused by Mr Demers.

A considerable concern is the increased risk of airway occlusion when a passive humidifier is used. In fact, one study[26] of the use of passive humidifiers was interrupted because of a fatal occlusion of the airway associated with the use of a passive humidifier. I conducted a meta-analysis of airway occlusion with active versus passive humidifiers (Fig. 4).[26–28,30,35–37] There was a large and significant increase in airway occlusion associated with the use of passive humidifiers. I submit that the increased risk of airway occlusion with a passive humidifier outweighs any benefit of reduced VAP.

A challenge with evidence-based medicine relates to how one takes the evidence from populations of patients and applies it to an individual patient. Here is my wish if I am the patient on the receiving end of the ETT: I will take my chances with a VAP, but please don't occlude my ETT. "*Let the Breather Beware!*"

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Reviews of Books and Other Media. Note to publishers: Send review copies of books, films, tapes, and software to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104.

Books, Films, Tapes, & Software

Bronchial Asthma: Principles of Diagnosis and Treatment, 4th edition. M Eric Gershwin MD and Timothy E Albertson MD PhD, Editors. Totowa, New Jersey: Humana Press. 2001. Hard cover, illustrated, 476 pages, $125.

Asthma is present in every patient population, with an increasing prevalence, and is ever-changing in relationship to our environment. The fourth edition of **Bronchial Asthma: Principles of Diagnosis and Treatment** holds fast to its title in that it is very specific to only asthma and does not stray into other pulmonary diseases. Drs Gershwin and Albertson have edited a great deal of information specific to asthma into an easily read text that covers asthma from the neonate to the adult. As mentioned in the preface, the target audience is the primary care physician. Specialists in asthma management will find this textbook comprehensive but occasionally spotty. The book would give an advanced respiratory therapist a better understanding of the diagnosis and treatment of asthma, but the paucity of inpatient management algorithms makes the book less useful for inpatient asthma caregivers.

The text is in 20 chapters, each of which is long enough to provide factual information regarding the subject matter but not so long as to overwhelm the reader, with the exception of Chapter 2, which deals with the pathogenesis of asthma. Chapter 2 extensively catalogs, discusses, and references all the current cellular, cytokine, and chemokine suspects in asthma pathogenesis—not light after-dinner reading! Even so, the key-point summary at the chapter's beginning helps the reader understand the context of what follows and an overview of current theory.

Chapter 3 discusses the clinical and allergy evaluation of asthma patients and focuses on the appropriate role of history, physical exam, pulmonary function, and atopic and possible serologic evaluations, distinguishing issues between small children and older patients with asthma. Details of pulmonary function testing are dealt with extensively in Chapter 4, through inclusion of charts, diagrams, tables, and illustrations that address the value and use of spirometry, lung volumes, and provocation testing. There are important clinical insights, particularly the value of FEV_1 assessment in asthma documentation. Sometimes being comprehensive is not as important as being clinically valuable!

Separate chapters deal with the differential diagnosis of asthma in children and adults, with the pediatric aspect being more organized and developed from the perspective of clinical frequency. By contrast in Chapter 6, which deals with the differential diagnosis of asthma in adults, asthma "look-alikes" are almost randomly presented. In addition, the distinction between intrinsic and extrinsic asthma invokes a nomenclature less often used today than in years past, as we have come to understand the complexity and overlap of different inflammatory processes giving rise to the clinical syndrome we now call asthma.

Chapters 7–10 are dedicated to treatment modalities. Each chapter discusses treatment for specific ages or clinical circumstances. However, sometimes recommendations dribble out at almost random intervals, such as in the section on treating adults with asthma, which is confusingly organized and presented. And though the role of inhaled corticosteroids is extensively covered, the recommendations imply that long-acting β agonists, such as salmeterol, may be used *before* the addition of inhaled anti-inflammatory steroids—an obviously flawed suggestion, according to the guidelines of the National Heart, Lung, and Blood Institute.[1] Curiously, no detailed discussion of status asthmaticus is presented, which limits the book's usefulness primarily to the outpatient setting. On the positive side, the very extensive and comprehensive discussion of asthma and pregnancy should be excellent reading for obstetrics professionals. But, on the negative side, the book references the 1993 consensus panel recommendation[2] of beclomethasone as the inhaled steroid of choice, which may lead some practitioners to the awkward choice of multiple puffs, multiple times per day, of a medication that most experts have relegated to the position of almost a historical curiosity. Most appropriate for those curious about what patients might be using without a physician's prescription is the extensive chapter on alternative therapies, which deals primarily with herbal and nonprescription treatments. It is unfortunate that what is also included in this chapter concerns alternative physician-prescribed treatments, including immunosuppressive medications; no references are given to buttress the statements made, such as regarding methotrexate's lack of efficacy and safety. Ideally an entire chapter would have been dedicated to dealing with refractory asthma and alternative therapies for that rare but clinically vexatious problem.

We commend the chapters dealing with special clinical problems, especially Chapter 12 on exercise-induced asthma. For the specialist dealing with this sometimes-difficult problem the discussion is illuminating and thoughtful. However, there is no discussion of sexuality and asthma—perhaps not to be considered minor by those afflicted. For some asthmatics the most strenuous activity is sexual intercourse, which may trigger a very frightening asthma attack. The discussions of allergic bronchopulmonary aspergillosis, aspirin sensitivity, anesthesia, and recreational drug use are well referenced and useful for all readers. Only in the chapter on anesthesia for asthmatic patients is there any substantial discussion of *status asthmaticus*. The comprehensive discussion (with extensive referencing) is welcomed on asthma topics such as occupational asthma and allergic bronchopulmonary aspergillosis, but, even so, some chapters emphasize conclusions from 20-year-old literature when discussing therapeutic options. For example, in the otherwise comprehensive discussion of allergic bronchopulmonary aspergillosis, inhaled corticosteroids are referenced as being of dubious use on the basis of studies performed in the 1970s, before the advent of more potent inhaled corticosteroids such as budesonide and fluticasone. And, too, the regimen presented for per os steroid taper is from 1979. Have no other recommendations been published since?

The final 3 chapters will assist the clinician in understanding the life modifications the asthma patient will deal with in living with asthma, the psychological impact of the disease, and the relationship of the care providers and the course they prescribe and

how these may impact them from a legal prospective.

The text in hard back is 464 pages, with a considerable index. Each chapter includes an extensive reference list. There are very few illustrations, mainly radiographs, but there are extensive tables and graphs in each chapter. The copy editing was very poor, however; numerous typographical errors litter the text, and unexplained abbreviations abound, although their meaning is usually evident. The most egregious editing error is on the first page of the chapter "Treatment of Asthma in Adults": the "key points" section is from a separate text on pain management; the section was apparently accidentally inserted at some point in the editing or paste-up process, and is unrelated to the chapter or the rest of the book.

In conclusion, **Bronchial Asthma: Principles of Diagnosis and Treatment** stands as an excellent text for students of respiratory disease to have on hand to address specific questions about asthma. For the most part it is comprehensive and all-inclusive. However, given today's Internet access to review articles that are more recent than many of the articles referenced within this text, the cost of $125 seems somewhat steep. But, on the whole, Sir William Osler's advice 100 years ago for his students to "read the texts" applies to this book in our time as well.

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Manual of Asthma Management. 2nd edition. Paul M O'Byrne MB and Neil C Thomson MD, Editors. London: WB Saunders. 2001. Soft cover, illustrated, 681 pages, $95.

The **Manual of Asthma Management** is a 681-page text comprising 5 sections and 52 chapters. The sections are: epidemiology and pathogenesis of asthma; diagnosis and investigations of asthma; assessment and treatment of asthma; management of asthma; and running an asthma service. As the section names suggest, the authors set out to cover nearly every aspect of asthma management. To accomplish this they recruited an impressive international panel of asthma authorities, which reads like a "Who's Who in Asthma," including Drs Barnes, Drazen, Holgate, Woolcock, Platts-Mills, Pauwels, and many others.

Much of this text targets physicians who are responsible for evaluating and managing asthma patients. As a reference, it contains material that appeals to physicians at all levels, from resident to pulmonary specialist. Having said that, it also functions well as a reference for respiratory therapists, nurses, and other providers who have contact with asthma patients, particularly in the chapters that deal with disease assessment and management, patient education, and special problems in asthma.

In the preface the editors state that they want to provide "comprehensive yet practical and easily accessed advice on the key issues of asthma management." It appears that they come close to accomplishing that objective. The large number of chapters (each by different authors) illustrates the editors' strategy of "splitting, not lumping" subjects, with chapter titles ranging from "Delivery Systems" to "Nocturnal Asthma." From this standpoint they create one or more homes for every category of asthma information, which caused some overlap in coverage of certain subjects (eg, management strategies) and some differences in the depth of coverage of topics from chapter to chapter. However, the material presented is accurate and well-referenced. A clear effort to support statements with evidence is seen throughout the manual. The 43-page chapter on "Management of Chronic Asthma in Children," for instance, is supported by 377 references.

The text is very reader-friendly in several respects. There is minimal use of pulmonary jargon, which illustrates the editors' desire to appeal to a broad readership. Tables and lists appear throughout the text to summarize important points, with key "take-home" points listed at the ends of most chapters. Each chapter is separated into intuitive subsections that allow the reader to quickly navigate to the subject of interest.

Although I think the majority of the manual is well-structured and well-written, several chapters stand out as particularly good reviews. The chapter on corticosteroids, by Peter Barnes, does an excellent job of explaining everything from mechanism of action to practical matters related to inhaled corticosteroid use. Several chapters very effectively address special situations in asthma, including occupational asthma, aspirin-induced asthma, and asthma in pregnancy. These chapters are very good summaries and are equally useful as a source of references for the pulmonary specialist or anyone who wishes to delve further into the subject. The chapter on management of chronic asthma in children is also notable for its thorough coverage of this broad subject.

Although my overall impression of the manual is very positive, there are a few shortfalls that should be pointed out. The text contains very few illustrations, which hinders its ability to educate the reader about certain complex subjects, such as the cascade of events that results in allergic airway inflammation and the multitude of changes in the airway that conspire to produce airway narrowing. The chapter on pulmonary function testing is heavily focused on theory, much of which is difficult to relate to asthma. I would like to see more detailed coverage in several areas, including the subject of remodeling in the airway and the recently-recognized importance of mucus in the generation and maintenance of airway obstruction. I think the chapters on patient education and compliance are too brief, given the critical importance of those factors in asthma management. On a related matter, minimal attention is given to alternative and complementary therapies in asthma, which are increasingly used by patients who are frustrated by inability to control the disease with medications. Finally, I would appreciate a separate chapter devoted to critical care, with special attention given to mechanical ventilation and anesthesia issues in the intubated asthmatic.

The international authorship should also be noted for American readers who are accustomed to more "North America-centric" medical writing. Much of the data presented are based on experience in the United Kingdom, with less emphasis on studies from the United States than is typically seen in publications of American origin. This is worth mentioning because there are some differences between the British *Guidelines*

on Asthma Management and the American guidelines, published by the National Asthma Education Prevention Program in 1997. Those differences are highlighted in the chapter on management of chronic asthma in adults.

In summary, I found the **Manual of Asthma Management** an excellent reference for this complex disease. It is well written, accurate, extensively referenced, and comprehensive. I was most impressed with the editors' ability to cover the subject in a manner that appeals to a broad readership. This manual contains something for nearly every health care provider who has contact with asthma patients, from patient educators to pulmonary specialists. I intend to keep it close at hand as an excellent overview of this complex and rapidly-changing subject.

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CRC Desk Reference for Allergy and Asthma. Hans-Uwe Simon MD et al. (CRC Desk Reference Series, Gerald Kerkut, Series Editor). New York: CRC Press, 2000. Hard cover, illustrated, 379 pages, $79.95.

The **CRC Desk Reference for Allergy and Asthma** is one in a series of medical science encyclopedias. I found the book's presentation unique for a medical reference, as there is no index and the topics are simply arranged in alphabetical order. The author's intention is to present each subject in such a concise manner that the reader will remember the important information presented. I typically read medical and science books by looking up certain subjects of interest in the index, in the manner described by this book's first author, Dr Hans-Uwe Simon, in the preface. Often I have to flip through many sections of related material before I find the subject I am interested in. The intention of this book is to give busy professionals the information they need quickly and concisely, and for the most part the authors achieved that goal. They also attempted to achieve a balance of providing enough information to be useful without providing so much that there is not sufficient time to read the subject matter.

Very important information is highlighted in boxes, with more extensive definitions provided in the remaining text. I found the highlighted text an exceptionally useful tool for conveying the most important information quickly. For instance, when discussing asthma treatment strategies, one section of highlighted text states, "Patients who do not settle on standard doses of inhaled corticosteroids (typically they will show persistent variability of peak expiratory flow, persistent nocturnal waking, or excessive use of a β_2 agonist) should be given a long-acting bronchodilator as well as their inhaled corticosteroids." That information could prove invaluable for the family practitioner managing a patient's asthma symptoms. In the text, italicized words are cross-referenced to other areas of the book. Although this is effective in keeping individual subjects short, I often found myself flipping back and forth when thoroughly investigating a topic.

The text covers basic immunology, clinical immunology, allergology, pharmacology, biochemistry, and molecular biology. There are simple definitions for most of the major respiratory diseases, such as chronic obstructive pulmonary disease, chronic bronchitis, and cystic fibrosis. The asthma material includes the definition, diagnosis, epidemiology, and treatment strategies, as well as prevention. I found the information very current and practical. As a critical care clinician I noticed that there is no information provided on managing acute severe asthma; however, I don't believe critical care clinicians are intended to be the majority of the audience for this text. There are 42 consecutive pages of asthma information as well as many cross-references to other areas of the book. I found the information on asthma pharmacology generally well presented and to include all the traditional medications, such as corticosteroids and theophyllines, as well as the newer ones, such as leukotriene receptor antagonists. There are basic definitions as well as very complete descriptions of their actions and reactions.

I did find some errors in the referencing to other sections. When looking up terbutaline the reader is sent to *β-agonists*, but there is no listing for *β-agonists*. I luckily stumbled across a listing for *β_2- agonists*, which referenced the term *agonists*, where I found the not-very-enlightening definition: "This group of drugs is particularly effective in the treatment of *Asthma* (see *asthma treatment strategies*)." This is one example of the authors not meeting their stated goal of providing quick and efficient information. The *β-agonists* information is presented further down on the same page. I would have been interested in some specific information on terbutaline, but no details are presented on the use of this drug. My comment may be biased by the critical care focus of my interests, as terbutaline is no longer frequently used in an outpatient setting.

I was also bothered by a contradiction in the text concerning the use of nebulizers to deliver medications. Page 46 reads: "Conventional inhalers may become ineffective because of complete airway obstruction by mucus plugs, and nebulized bronchodilators (see *nebulizer*) are useful to ensure that the drugs are delivered to all ventilated airways." Here the authors appear to be suggesting that nebulizers are superior to metered-dose inhalers and that a nebulizer can somehow make a completely obstructed mucus-plugged airway a ventilated airway that will allow drug deposition. Then on Page 333, when defining spacer devices, an opposing, strongly worded, and correct statement is made that: "Spacer devices [ie, metered-dose inhalers] have been shown to be as effective as *nebulizers* in the treatment of acute severe asthma."

There are 94 tables and 50 figures, the majority of which are simple, clear, and concise. Unfortunately, the flow-volume loop, which is so frequently used to diagnose air flow obstruction, is not graphically depicted. Although there are basic descriptions of the loop and other lung function tests and their diagnostic values, a figure would have been valuable. The sections on occupational asthma, dermatitis, and rhinitis include diagnosis, therapy, economic impact, and prevention. Table 74 is an excellent breakdown of the allergens and professions that are frequently associated with occupational asthma.

Appropriately, a large portion of the text deals with allergic diseases and allergic inflammation. It provides complex information on actions and reactions at the cellular level, much of it graphically illustrated. How to properly place and read a patch test is clearly described and is an excellent practical inclusion in the text. There is an in-depth review (9 pages) on mast cell morphology, mediators, activation, receptors, and stabilizers. Table 68 in this section is particularly valuable in that it describes some important mast cell mediators and their role in asthma. There is extensive information

provided on potential allergens such as house dust mites, molds, pollens, animal dander, and environmental contaminants. Specific food allergies are covered, as well as the latex-fruit syndrome. I found the inclusion of latex allergy information current and timely in that "Health care workers have a 5–10% prevalence of latex allergy." Identifying and treating latex allergy has quickly become a large problem for all types of clinicians, as well as a patient safety issue.

The authors are an impressive list of experts from throughout Europe, with a minority of the contributors hailing from the United States, which gave the text a distinctly European flavor. One example of this is that the normal ranges for P_{aCO_2} and P_{aO_2} are only provided in kilopascals; no conversions to mm Hg are provided. The term *rhonchi* is defined as an "old-fashioned term for wheezes." One problem in my copy of the book is not related to the authors: pages 290, 291, 294, 295 are unreadable because the outer third of the printed text did not transfer to the pages.

The intended audience is primarily scientists, researchers, and students whose primary interest is allergy and asthma. Practicing clinicians such as pulmonologists, general practitioners, pediatricians, allergists, pulmonary nurses, and respiratory therapists will all find useful, up-to-date information pertinent to their daily practice. I would caution that this is an intensely scientific text and the majority of the information concerns allergic inflammation at the cellular level. It is very heavily weighted toward immunology, molecular biology, and biochemistry. If you are a clinician whose primary interests are asthma diagnosis, management, and treatment you may want to consider a different text.

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Allergies and Asthma for Dummies. William E Berger MD. Foster City, California: IDG Books Worldwide. 2000. Soft cover, illustrated, 399 pages, $19.99.

Allergies and Asthma for Dummies is promoted as an "all you need to know about asthma and allergies" reference book written specifically for allergy and asthma patients and their loved ones who care for them. As I began to read, I questioned how such a broad and complicated subject matter could possibly be covered in an easily understandable way for the layperson.

My professional background includes over 20 years as a respiratory therapist, and the majority of my experience has been working at a regional trauma and teaching center. On reflection I realized that my knowledge extends beyond the professional setting. I know first hand what it means to be an allergy and asthma sufferer with family members, including children, who have experienced the extreme symptoms of allergy and asthma, including exercise-induced asthma.

The paperback cover catches the reader's eye; it comes in the easily identifiable black and yellow style of all of the "For Dummies" books. The "Dummies" style continues when you open the book, with a "cheat sheet" that includes "Dr Berger's Top Ten List of Allergy Triggers," and the book ends with "Ten Famous Folks with Asthma," including Augustus Caesar and Jackie Joyner-Kersee. Illustrations and cartoons help explain concepts, including basic anatomy, pathophysiology, and related teaching points.

The book is organized to guide the reader to information of immediate interest without having to read the entire book; it includes a comprehensive index. Dr Berger does an exceptional job in not bogging the reader down with details they don't want or need. He does this with "Dummies"-style icons warning the reader of "technical stuff" and "medicalese" (medical terminology). This gives the reader a chance to skip or dive into the more difficult terminology.

Dr Berger covers the topics of allergies and asthma from prevention to life-threatening emergencies. Included are "Getting the Most from Your Doctor Appointment" and "How to Be a Knowledgeable Consumer." He gives examples of what information to have available and what questions to ask the physician. The author is meticulous in his attention to detail when he includes the importance of choosing an insurance company. He cautions against looking only at the cost and encourages researching what the insurance company offers and how long it may take to get medical care. This information is vital to patients of lower income, for whom understanding that paying more now may mean paying less overall *and* that they are more likely to get medical care before the condition becomes life threatening and requires hospitalization—a philosophy not always followed by some low-cost medical insurance plans.

The author doesn't miss an opportunity to catch the reader's attention by exposing common myths and directing the reader immediately to chapters that discuss relevant facts. Chapter 1 begins by explaining how allergies can affect many organs throughout the body, including the lungs. Chapter 2 delves into the immunologic basis of allergic reactions, going into complicated detail that includes abnormal immune reactions. Dr Berger does an exceptional job with his clear and simple style and use of illustrations, giving just enough detail not to overwhelm the reader. However, there is no way of getting around some complicated terminology foreign to most patients. If the reader chooses to dive into that information it is there and written as simply as possible.

Chapter 3 discusses how to deal with doctor visits and insurance companies, and I am impressed by Dr Berger's level of honesty and detail on this subject.

Chapter 4 includes diagnosis and treatment of hay fever. This chapter also reminds the patient of his or her responsibility for keeping track of the timing of and events that trigger allergies, so the physician can determine what is causing the allergic reaction. Chapter 5 deals with pollens, molds, and dust and their role as triggers. Chapter 6 helps the reader understand the importance of avoiding triggers and describes strategies of how to manage allergens and irritants, including the importance of keeping the house dust-free. Except for the compulsive minority, keeping a house dust-free is low on most people's priority list. Fortunately, Dr Berger explains the whys and hows of practical dust-free living for allergic readers. Chapter 7, "Relieving Your Rhinitis," illuminates the complicated subject of pharmacology as related to rhinitis. Chapter 8 continues with skin tests and their indications. Dr Berger explains the goals of immunotherapy and includes factors that may help the patient determine if immunotherapy is an option to be discussed with the physician. The chapter concludes with a thorough description of the treatment the patient would have to comply with, as well as possible adverse effects—information that is vital to such a decision. Chapter 9 provides information to help the reader avoid allergy complications such as sinusitis and ear infections.

In Chapters 10–15 Dr Berger thoroughly discusses the fact that asthma is often the

result of underlying chronic inflammation in the airways, which can result in airway remodeling—a condition that often results in lung scarring and irreversible lung damage. In Chapter 11 readers are lead to identify what triggers their asthma and how to manage those triggers. Chapter 12 explains the pharmacologic aspect of asthma therapy. Dr Berger discusses the goals of therapy, the types of medication and how they work, and the various drug delivery systems. He explains very clearly the importance of consistently taking medications as planned, even during asymptomatic periods. Tables provide quick comparisons of short-term and long-term control medications. Chapter 13 discusses long-term management of asthma, emphasizing a team approach that includes the patient, family, and health care professionals. Chapter 14, "Asthma and Allergies during Childhood," discusses the signs and symptoms of asthma in children, which can be complicated because children may not be able to communicate and the caregiver must be sensitive to the manifestations of this potentially life-threatening disease. Chapter 15 discusses the special issues of allergy and asthma control during pregnancy.

Part IV (Chapters 16–18) covers allergic skin conditions and is very informative and thorough. Chapter 16 discusses atopic dermatitis, from understanding what it is to prevention and management of the condition. Chapter 17 is on allergic reactions from skin contact, and explains how to differentiate between reactions caused by an "irritant" and an "allergen." Chapter 18 discusses why taking hives and angioedema seriously may save your life and discusses management by avoiding triggers and medication used to prevent and treat the condition.

Dr Berger continues the subject of allergy cause, management, and treatment in Part V, "Food, Drug, and Insect Reactions: A Really Bad Trip." Each topic section (food, drug, and insect reactions) helps the reader understand the cause, management, and treatment of the allergy. The book concludes with Part VI, "The Part of Tens," which includes 2 chapters: "Ten Tips for Traveling with Allergies and Asthma," and "Ten Famous Folks with Asthma."

If you subscribe to the philosophy that "when the student is ready to learn, the teacher will appear," this book is a winner. It covers technical information very well, with a writing style that is anything but dry. Still, however simply written, some of this information will be difficult going for the average reader. Dr Berger's welcome use of humor helps hold the reader's interest, but the reader will need to be motivated and focused to understand the technical information.

I would not recommend this book to the patient who is not motivated to learn about the disease. It will most benefit those who want to be involved in the decisions regarding their own health. This book provides an exceptional amount of up-to-date information for the patient and those who share in care, including the health care professional.

Although intended for the patient and family, I find this book an exceptional reference for the patient educator. Education is often the key to patient compliance with medical instruction. Once a professional relationship with the patient is established, the information and teaching techniques Dr Berger uses could be useful in developing an approach to the education of even reluctant patients. Overall the book is entertaining and informative and is a must read for any professional working in asthma education.

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Control Your Child's Asthma: A Breakthrough Program for the Treatment and Management of Childhood Asthma. Harold J Farber MD and Michael Boyette. New York: Henry Holt and Company. 2001. Soft cover, illustrated, 231 pages, $14.

In the last decade, much has been learned about asthma and its management. Therefore, a book such as **Control Your Child's Asthma** would appear to fill an important need as a resource for parents of asthmatic children. This book is the latest of several such manuals available to help families expand their knowledge of a disorder that can in most cases be controlled with available treatments. However, often such attempts at control fall short because physicians and other caregivers are not given sufficient time with their patients to explain the chronic nature of asthma and the importance of developing and adhering to the child's asthma treatment plan. If read and referred to often, a book such as this will greatly improve outcomes.

The book is a product of medical personnel at the Kaiser Medical Group and their experiences over the past 2 decades. Although some may object to the many references to their system of health care, the book should not be considered promotional and is filled with valuable information. Such a book often reflects the experiences and practices of its authors; therefore, most physicians will find errors and differences of opinions. Such was the case when I first read the book, but fortunately all my objections were minor and inconsequential overall (for example, Accolate needs to be taken with food). If there is an objection to be made about the book, it would be about the subtitle, "A Breakthrough Program for the Treatment and Management of Childhood Asthma," which might lead some readers to believe that there is something written in this book that is unique about asthma control and that cannot be obtained elsewhere, which is not the case: most specialists who see asthmatic children agree on what should be done. It is patient and family treatment-program adherence that often can be improved with books such as this. This book is well written, organized, resourceful, and filled with answers to many questions, some not so commonly asked.

The book is organized into 3 parts: "The Asthma Revolution," "The Three Lines of Defense," and "Parents' Guide." In addition, for parents and patients who desire to "go to the source" for information, there are references to medical articles on key topics.

In "Three Lines of Defense" the authors describe the foundation for controlling asthma: (1) Manage the environment, (2) manage the bronchial tubes, and (3) manage the flare-ups. This well-organized section helps the family develop a management plan for asthma that can, if followed, lead to better control. No one strategy is emphasized more that the other (which is appropriate), and the section gives detailed information on treatment, reasonable expectations for success, and guidelines on managing exacerbations.

In the chapter on managing the environment, various asthma triggers are discussed in appropriate detail, and the importance of avoidance measures is emphasized. It should be clear to the reader that therapy starts here. In the chapter on managing the bronchial tubes the emphasis is on the variety of medications available, their proper role in treatment, and their proper use. Since control of this disorder often can come only through

the proper selection of a medication to treat the underlying inflammation process, this chapter should help to greatly improve control through a better understanding of risk/benefit ratios. Fear of adverse effects should not be part of a good management program, but medical personnel often minimize the impact that fear has on parents and patients, and this book addresses the issue openly and thoroughly. Since many asthma medications need to be inhaled correctly to be maximally effective, I think this chapter will be particularly helpful to the many patients who have difficulty achieving proper inhaler technique. In the chapter on managing flare-ups, the emphasis is on early detection and implementing changes sooner rather than later, whether at home or school. This chapter also can be extremely helpful in preventing the serious outcomes that often result from delays. The stepwise approach outlined here is appropriate and does not suggest a delay in obtaining medical care.

The third part of the book, "Parents' Guide," is filled with information on finding the right doctor or health plan and properly using the various inhalers and peak flow devices. There are chapters on infants and toddlers, teenagers, and special needs outside the home (at school and with family and friends), as well as on complementary and alternative treatment, to which many turn for answers without receiving an honest or complete answer. Finally, potential future therapies are discussed briefly but with enough detail to inform without raising unrealistic expectations. The answers to the 17 frequently asked questions, which are covered thoroughly at the end, will, for many, be worth the price of the book.

This book, if read and followed by all parents of asthmatic children, would indeed fulfill the promise of its title, "Control Your Child's Asthma."

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Environmental Asthma. Robert K Bush MD, Editor. (Lung Biology in Health and Disease, Volume 153, Claude Lenfant, Executive Editor.) New York: Marcel Dekker, 2001. Hard cover, illustrated, 370 pages, $165.

Environmental Asthma is the 153rd volume of the Lung Biology in Health and Disease series. In 370 pages, the editor, Robert Bush, incorporates 15 chapters focusing on how environmental factors influence the onset and clinical expression of asthma. In an opening chapter Dr Bush identifies the themes that the remainder of the book expands on, and the last chapter takes a forward-looking approach. The first and last chapters give the reader some insight into Dr Bush's concept of environmental asthma and they represent valuable summaries. Of all the common respiratory diseases, asthma is the one in which there are clear environmental links involving multiple factors. Thus, the first 4 chapters sequentially deal with antigen-induced airway disease, with specific focus on dust mites, animal allergens, funguses, and pollens. These chapters provide reasonable overviews of rapidly expanding fields, each of which could be the topic for a single volume! The advent of molecular biology has provided fascinating insights into allergen structure in relation to function. In this context, more detail would have been helpful on what makes an allergen an allergen, how the intrinsic biological activities of some allergens may confer a sensitization advantage, and how allergens are processed at the airway surface and subsequently translated into a systemic immune response.

Since the book deals with the environmental aspects of asthma, the following 3 chapters on air pollution and the indoor and outdoor environments contain first-class descriptions of additional key factors other than allergen exposure that are believed to be important in the onset and progression of asthma. Although not explicitly stated, it is implied that asthma arises from a sequential progression in susceptible individuals: allergen exposure → sensitization → mediator release → inflammation → altered airway function. However, what is not brought into this sequence is *how* air pollution, exposure to environmental tobacco smoke, viral infections, and dietary risk factors (eg, low antioxidants) could influence airway response to allergens. Since the population-attributable risk of atopy in asthma is only 35–40%, these additional factors are important and need to be put into context. The indoor environment is given special attention, quite rightly, because most humans spend the greatest part of their time indoors; but, when assessing the influence of the environment on disease expression, total personal exposure as well as consideration of troughs and peaks of exposure and co-exposures become important issues to which little attention is directed. The 3 chapters on occupational asthma are most informative, the first containing a comprehensive list of occupational sensitizing agents. The well-informed discussion on pathophysiological mechanisms of occupational asthma makes these 3 chapters especially valuable.

Chapter 14 provides a concise overview of the use of immunotherapy in asthma management and the recent trials of novel delivery routes, such as sublingual and nasal. What also would be valuable is a detailed appraisal of allergen reduction strategies in primary, secondary, and tertiary prevention.

Overall, this book provides a reasonable overview of the role of environmental exposures in asthma, though many of the subjects are dealt with rather superficially—much of this information can be found in current major textbooks. Thus an opportunity was missed to deal with current issues of gene-environment interaction by appraising the modern immunology literature in relation to the early-life origin of asthma and how that might be connected (or not) to other organ manifestations of allergy, the literature on the rising trends in allergy and asthma as they affect the developed and developing world and possible underlying mechanisms (eg, a thorough appraisal of the "hygiene hypothesis"), and the influence of the environment on the natural history of asthma and the role of airway wall remodeling. Important environmental factors are also excluded, such as the role of viral (and some bacterial) infections and diet (especially the influence of obesity and anti-oxidants). Of the allergens that are tightly linked to asthma, cockroaches and a wide range of antigens that depend on climatic and housing conditions in different parts of the world are dealt with only fleetingly. Finally, the text has a somewhat "dated" feel about it because it does not take advantage of the extensive cell and molecular biology literature available in this field and work that knowledge into the sections dealing with pathophysiology and future treatment.

Though this book provides a readable overview of environmental factors in asthma, when compared to other books in this series, its treatment of the topic is somewhat superficial and narrow. The book lacks illustrations but, as a textbook, at $165, it would be a good read for clinicians and scientists who wish to obtain an overview

of this subject without demanding too much effort.

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Cilia and Mucus: From Development to Respiratory Defense. Matthias Salathe, Editor. New York: Marcel Dekker. 2001. Hard cover, illustrated, 426 pages. $185.

This book contains the conference proceedings from the second International Meeting on Mucus and Cilia, held in Italy in 1999, at which clinicians and scientists shared and discussed their findings on the biology of the mucociliary apparatus. The contributors are leaders in their fields of research, and I commend the editor for organizing such a heterogeneous group. The proceedings of the first International Meeting on Cilia, Mucus, and Mucociliary Interactions, held in Jerusalem in 1997, were summarized in the book *Cilia, Mucus and Mucociliary Interactions*, edited by GL Baum, Z Priel, Y Roth, N Liron, and EJ Ostfeld, and published by Marcel Dekker in 1998.

Cilia and Mucus: From Development to Respiratory Defense is in 3 parts: cilia, mucus, and mucociliary interactions. Each part begins with a comprehensive introduction that sets the research in its clinical context. The chapters are structured as research papers and many include results from the writers' own research.

Part I (Chapters 1–15) discuss the essentials of cilia physiology. Chapters 1 and 2 present the structure of the cilia, Chapters 3–8 examine the regulation of ciliary beat frequency by Ca^{2+}, Na^{+}, and the guanosine-monophosphate (GMP)-dependent protein kinase. Chapters 9–11 discuss the genomics of primary ciliary dyskinesia. Chapters 12–15 introduce in vitro models for studying mucociliary function. Chapters 13 and 14 describe the clinical relevance of the ciliary beat frequency changes due to *Streptococcus pneumoniae* and macrolide antibiotics.

Part II (Chapters 16–25) focuses on airway secretions. Chapter 16 reviews mucins biology relevant to the lung. Chapters 17–19 explain the role of myristoylated alanine-rich protein kinase C-substrate (MARCKS) and human neutrophil elastase on mucin gene expression and protein production. Chapter 21 describes the discovery that the MUC1 mucin is a receptor for *Pseudomonas aeruginosa*. In Chapter 24 it is hypothesized that mucins might be useful in the diagnosis of malignant pleural effusions. Chapter 25 describes the potential use of charged oligosaccharides for mucolytic therapy.

Part III (Chapters 26–35) describes mucociliary interactions and places the first 2 parts of the book in a more clinically relevant context. Chapters 26–30 discuss the physiology of mucociliary interactions and describe techniques to measure clearance in vivo. Chapters 31–35 then apply those techniques to asses the effect of mannitol, neutrophil elastase, and cough on mucociliary clearance.

The primary strength of this book is that it provides comprehensive updates and summaries of this field of research. Because the chapters are fairly short, the material is sometimes presented a bit superficially, but there are many well done figures and tables. Because the meeting the book commemorates was held in the spring of 1999, some of the chapters are out of date, but this is still the most comprehensive and recent reference in this field. The book is possibly too detailed for the clinician but it is a must for researchers. It gives a good overview of some of the most important aspects of mucus and cilia and their interaction, and it also describes the clinically relevant relationships.

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AARC Clinical Practice Guideline

Selection of an Oxygen Delivery Device for Neonatal and Pediatric Patients — 2002 Revision & Update

NPODD 1.0 PROCEDURE:
The selection of an oxygen delivery system for neonatal and pediatric patients includes patients with and without artificial airways.

NPODD 2.0 DESCRIPTION:
The administration of supplemental oxygen to neonatal and pediatric patients requires the selection of an oxygen delivery system that suits the patient's size, needs, and the therapeutic goals. Oxygen delivery systems are categorized as either low-flow (variable performance) or high-flow (fixed performance) systems.[1] Low-flow provide an F_{DO_2} (fractional concentration of delivered oxygen) that varies with the patient's inspiratory flow and are classified as variable-performance oxygen delivery systems.[1] High-flow systems can provide a specific F_{DO_2} at flows that meet or exceed the patient's inspiratory flow requirement and are classified as fixed-performance oxygen delivery systems.

2.1 Low-flow systems:

2.1.1 Nasal cannulas consist of two soft prongs that arise from oxygen supply tubing.[2] The prongs are inserted into the patient's anterior nares, and the tubing is secured to the patient's face. Oxygen flows from the cannula into the patient's nasopharynx, which acts as an anatomic reservoir.[3] The fractional concentration of inspired oxygen (F_{IO_2}) varies with the patient's inspiratory flow.[4]

2.1.2 Nasopharyngeal catheters are soft tubes with several distal holes. The catheter should be inserted into the patient's nose to a depth equal to the distance from the ala nasi to the tragus[5] or be gently advanced and then withdrawn until it rests slightly behind the uvula.[1] The tube, secured to the patient's face, is connected to oxygen supply tubing. Oxygen flows from the catheter into the patient's oropharynx, which acts as an anatomic reservoir.[6] The F_{IO_2} varies with the patient's inspiratory flow.[1]

2.1.3 Tracheostomy oxygen adapters are devices that attach either directly to a tracheostomy tube or to a heat-moisture exchanger (HME), which is then attached to the tube. (HMEs or artificial noses collects a patient's expired heat and moisture and returns it during the following inspiration.[7]) The oxygen supply tube connected to the adapter provides a blow-by source of oxygen that results in a variable F_{IO_2}. These devices are intended for short periods such as brief transports or to increase patient mobility.[8]

2.1.4 Transtracheal catheters are devices that delivery gas directly into the trachea via a small percutaneous catheter held in place with a bead chain necklace.[9]

2.2 Reservoir systems:

2.2.1 Simple oxygen masks are plastic reservoirs designed to fit over the patient's nose and mouth and be secured around the patient's head by an elastic strap.[10] An increased reservoir effect is produced by adding the volume of the mask.[1] Oxygen is delivered through a small-bore tube connected to the base of the mask. Holes on each side of the mask provide an egress for exhaled gases and serve as room-air entrainment ports.[10] The F_{IO_2} varies with the patient's inspiratory flow, mask fit and patient respiratory pattern.[1,11]

2.2.2 Partial-rebreathing masks are similar to simple oxygen masks but contain a reservoir at the base of the mask.[10] The reservoir receives fresh gas plus exhaled gas approximately equal to the volume of the patient's anatomic dead space.[10] The

oxygen concentration of the exhaled gases combined with the supply of fresh oxygen, permits the use of flows lower than those necessary for other devices (eg, non-rebreathing masks), and potentially conserves oxygen use.

2.2.3 Non-rebreathing masks are similar to partial-rebreathing masks but do not permit the mixing of exhaled gases with the fresh gas supply.[10] A series of one-way valves ensures a fresh oxygen supply with minimal dilution from the entrainment of room air.[1] The one-way valve over the reservoir bag prevents entry of expired gas, and the one-way valve over one of the side ports limits entrainment of room air.[1] This design provides a higher F_{IO_2} than the simple and partial-rebreathing masks and the nasal devices providing the mask fits correctly.[1]

2.3 High-flow systems:

2.3.1 An air-entrainment mask contains a jet orifice and air entrainment ports and is designed to fit over the patient's nose and mouth and is connected to oxygen supply tubing.[1] Oxygen under pressure is forced through a small jet orifice entering the mask. The velocity increases causing a shearing effect distal to the jet orifice, which causes room air to be entrained into the mask.[12] The total flow provided by the mask is determined by the cross-sectional area of the entrainment ports, the diameter of the jet orifice, and the oxygen flow to the jet. The F_{DO_2} is determined by the dimensions of the jet and the entrainment ports. The entrainment mechanism is based on the principles described by Bernoulli.[12] A collar can be attached to the base of the corrugated hose for supplemental humidification,[13] and the device can be adapted to a tracheostomy collar.

2.3.2 Air-entrainment nebulizers are gas-powered, large-volume nebulizers that contain an adjustable air-entrainment port, which determines specific oxygen concentrations.[10,14] In addition to providing particulate water with or without added medication, heated nebulizers can deliver gas saturated with water vapor at body temperature.[15] A corrugated hose serves as a conduit from the nebulizer to an aerosol mask, face tent, tracheostomy collar, or T-piece.[13]

2.4 Enclosure Systems:

2.4.1 Oxygen hoods are transparent enclosures designed to surround the head of the neonate or small infant.[10,16] A continuous flow of humidified oxygen is supplied to the hood.[10,16] Transparent enclosures in larger sizes (so-called tent houses or huts) are available for patients who are too big for neonatal-size hoods.[13]

2.4.2 Closed incubators are transparent enclosures that provide a warm environment for small infants with temperature instability.[10] Supplemental oxygen can be added to incubators but may result in an increased oxygen concentration.[10] The primary purpose of an incubator is to provide a temperature-controlled environment. Humidification is available through a baffled blow-over water reservoir; however, due to the high risk of infection associated with this humidification system, alternative sources are used.[10] Therefore, the incubator is not further discussed as an oxygen delivery device.[13]

NPODD 3.0 SETTING:

Oxygen delivery devices are used in a number of settings including hospitals, clinics, extended care facilities, the home, and patient transport vehicles.

NPODD 4.0 INDICATIONS:

The selection of an oxygen delivery device is indicated with:

4.1 documented hypoxemia[17,18]

4.2 an acute situation in which hypoxemia is suspected or in which suspected regional hypoxia may respond to an increase in P_{aO_2}. Substantiation of P_{aO_2} is required within an appropriate period of time following initiation of therapy.[17,18]

NPODD 5.0 CONTRAINDICATIONS:

5.1 No specific contraindications to delivering oxygen exist when indications are judged to be present.[18]

5.2 Nasal cannulas and nasopharyngeal

catheters are contraindicated in patients with nasal obstruction (eg, nasal polyps, choanal atresia, etc).[10]

5.3 Nasopharyngeal catheters are contraindicated in the presence of maxillofacial trauma,[19] in patients in whom a basal skull fracture is present or suspected,[18] or coagulation problems exist.[1]

5.3.1 It is the expert opinion of the Clinical Practice Guideline Steering Committee (2002) that nasopharyngeal catheters are not appropriate for oxygen administration in the neonatal population.

5.4 Although opinions vary,[21-24] infants intubated for airway protection should probably be placed on CPAP (ie, physiologic CPAP) for supplemental oxygen rather than on a T-piece because of the loss of physiologic end-expiratory pressure created by an open glottis.

NPODD 6.0 HAZARDS/PRECAUTIONS/POSSIBLE COMPLICATIONS:

6.1 Physiologic:

6.1.1 The etiology of retinopathy of prematurity, especially the role of oxygen, is controversial. Care should be taken when supplemental oxygen is provided to preterm infants (< 37 weeks gestation). It is suggested that oxygen supplementation should not result in a $P_{aO_2} > 80$ torr.[25]

6.1.2 The administration of supplemental oxygen to patients with certain congenital heart lesions (eg, hypoplastic left-heart, single ventricle) may cause an increase in alveolar oxygen tension and compromise the balance between pulmonary and systemic blood flow.[26-28]

6.1.3 The administration of supplemental oxygen to patients suffering from paraquat poisoning or to patients receiving certain chemotherapeutic agents (eg, bleomycin) may result in pulmonary complications (eg, oxygen toxicity and pulmonary fibrosis).[29,30]

6.1.4 Stimulation of the superior laryngeal nerves may cause alterations in respiratory pattern if the gas flow from the oxygen source is cool and is directed at the face of the infant.[31]

6.1.5 Inappropriate selection of F_{DO_2} or oxygen flow may result in hypoxemia or hyperoxemia.

6.2 Equipment-related

6.2.1 Nasal cannulas:

6.2.1.1 Skin irritation can result from material used to secure the cannula[3,32] or from local allergic reaction to polyvinyl chloride.[33]

6.2.1.2 Improper sizing can lead to nasal obstruction or irritation.[3,34]

6.2.1.3 Displacement can lead to loss of oxygen delivery.[3]

6.2.1.4 Inadvertent CPAP may be administered depending upon the size of the nasal cannula, the gas flow, and the infant's anatomy.[35-37]

6.2.1.5 Irritation can result if flows are excessive.

6.2.2 Nasopharyngeal catheters:

6.2.2.1 Improper insertion can cause gagging[6] and nasal or pharyngeal trauma.[6]

6.2.2.2 Improper sizing can lead to nasal obstruction or irritation.[3,34]

6.2.2.3 Excessive flow can produce pain in the frontal sinuses.[10]

6.2.2.4 Pneumocephalus is a rare but possible complication.[38]

6.2.2.4 Excessive secretions and/or mucosal inflammation can result.[5,39]

6.2.2.5 Skin irritation may result from material used to secure the cannula[3,32] and/or from local allergic reaction to polyvinyl chloride.[33]

6.2.2.6 Occlusion of distal openings may occur.[1]

6.2.2.7 Excessive flow may cause gastric distention.[1]

6.2.3 Transtracheal catheters:

6.2.3.1 Increase risk of infection compared to nasal cannulas and catheters[40,41]

6.2.3.2 Increased risk of complications[42-44]

6.2.4 Masks:

6.2.4.1 Aspiration of vomitus may be more likely when a mask is in place.

6.2.4.2 Irritation may result from tight application.[10,45]

6.2.4.3 Rebreathing of CO_2 may occur if total O_2 flow is inadequate.[14,46]

6.2.4.4 It is the expert opinion of the Clinical Practice Guideline Steering Committee (2002) that partial rebreathers or non-rebreathers are not appropriate for the neonatal population.

6.2.5 Air-entrainment nebulizers:

6.2.5.1 produce high noise levels in enclosed environments (eg, hoods, incubators) and may induce hearing impairment;[47] when an air-entrainment nebulizer is used in an enclosed environment, the entrainment port should be set on 100% (ie, closed) and the nebulizer powered either by a blender or by compressed air with titration of oxygen to the desired concentration.[13]

6.2.5.2 are susceptible to contamination;[48-50]

6.2.5.3 may cause bronchoreactivity in patients with reactive airways when used with nonisotonic solutions;[51]

6.2.5.4 may create unwanted torque and increase the likelihood of inadvertent extubation or decannulation of the patient when used with a T-piece and applied directly to an endotracheal or tracheostomy tube;[13]

6.2.5.5 may not provide particles of desired size range and in a predictable dose;[52]

6.2.5.6 if unheated, may induce cold stress in neonates;[31,53]

6.2.5.7 Condensate in tubing may result in advertent lavage when attached to the endotracheal tube.

6.2.6 Hoods and transparent enclosures:

6.2.6.1 Prolonged exposure to humidified oxygen may increase risk for cutaneous fungal infection.[54]

6.2.6.2 Inadequate or loss of gas flow may result in hypoxia or hypercapnia.

6.2.6.3 Temperature within enclosures should be closely monitored to reduce the potential for cold stress or apnea from overheating in neonates.[31,53]

6.2.6.4 Use of an improperly sized hood can result in irritation of the infant's skin.[13]

6.2.7 Tracheostomy oxygen adapters: Adapters may create unwanted torque and increase the likelihood of inadvertent decannulation of the patient, and HMEs may increase work of breathing to an unacceptable level in patients < 8 kg if dead space and resistance are high.[55]

6.3 During laser bronchoscopy, minimal levels of supplemental oxygen should be used to decrease the risk of intratracheal ignition.[18,56]

6.4 Fire hazard is increased in the presence of increased oxygen concentrations.[18]

6.5 Bacterial contamination has been associated with certain nebulization and humidification systems.[48-50]

NPODD 7.0 LIMITATIONS:

7.1 Nasal cannulas:

7.1.1 Changes in minute ventilation and inspiratory flow affect air entrainment and result in fluctuations in F_{IO_2}.[57-59]

7.1.2 Prongs are difficult to keep in position, particularly with small infants.[3,32,60]

7.1.3 The effect of mouth versus nose breathing on F_{IO_2} remains controversial.[61-64]

7.1.4 Use may be limited by the presence of excessive mucus drainage, mucosal edema, or a deviated septum.[10]

7.1.5 Maximum flow should be limited to 2 L/min in infants and newborns.[35,57,58]

7.1.6 Care should be taken to keep the cannula tubing and straps away from the neck to prevent airway obstruction in infants.

7.1.7 Discrepancies between set and delivered flow can occur in the same flowmeter at different settings and among different flowmeters.

7.1.8 Discrepancies in flow and oxygen concentration between set and delivered values can occur in low-flow blenders at flows below the recommended range of the blender.

7.2 Nasopharyngeal catheters:

7.2.1 Method is in less common use because of the complexity of care.[34]

7.2.2 F_{IO_2} is difficult to control and measure.[57,58]

7.2.3 Effect of mouth versus nose breathing on F_{IO_2} remains controversial.[61-64]

7.2.4 Use may be limited by excessive mucus drainage, mucosal edema, or the

presence of a deviated septum.[6]

7.2.5 Catheter should be cleared frequently to prevent occlusion of the distal holes.[5,6] The patient should be observed for evidence of catheter occlusion, and the catheter should be alternated between nares every 8-12 hours and changed daily.[65]

7.2.6 Catheter sizes less than 8 Fr are less effective in oxygen delivery.[5]

7.2.7 Lower oxygen concentrations are delivered if the catheter is placed in the nose rather than in the pharynx.[5]

7.2.8 Low-flow flowmeters (< 3 L/min) should be used.[5]

7.2.9 Discrepancies between set and delivered flow can occur in the same flowmeter at different settings and among different flowmeters.

7.2.10 Discrepancies in flow and oxygen concentration between set and delivered values can occur in low-flow blenders at flows below those recommended by the manufacturer.

7.3 Transtracheal catheters:

7.3.1 Method is in less common use because of the complexity of care.[1]

7.3.2 Requires frequent medical monitoring[1]

7.3.3 Replacement catheters are costly.[1]

7.3.4 Increased time needed for candidate evaluation and teaching[1]

7.4 Masks:

7.4.1 provide variable F_{IO_2} depending on inspiratory flow and construction of the mask's reservoir[13] and are not recommended when precise concentrations are required;[66,67]

7.4.2 are confining and may not be well tolerated;[68]

7.4.3 interfere with feeding;[6]

7.4.4 may not be available in sizes appropriate for all patients;

7.4.5 require a minimum flow per manufacturer's instructions to avoid possible rebreathing of CO_2.[14,31,46]

7.4.6 The maximum F_{IO_2} attainable with a simple, non-rebreathing or partial-rebreathing mask in neonates, infants, and children has not been well documented.[46]

7.4.7 The performance of air-entrainment masks may be altered by resistance to flow distal to the restricted orifice (resulting in higher F_{DO_2} and lower total flow delivered). The total flow from air-entrainment masks at settings greater than 0.40 may not equal or exceed the patient's inspiratory flow.[10,18]

7.4.8 Performance is altered if the entrainment ports are blocked.

7.5 Air-entrainment nebulizers:

7.5.1 are vulnerable to alterations described in Section 7.4.7;

7.5.2 should have temperature monitored if they are heated. (Cool mist is not recommended for newborns because of the potential for cold stress.[31,53]) In newborns, the temperature of the gas-aerosol mixture at the patient should be approximately equal to the desired environmental temperature.[53]

7.5.3 may have performance altered by resistance to flow distal to the restricted orifice (resulting in higher F_{DO_2} and lower total flow delivered). The total flow from air-entrainment nebulizers at settings greater than 0.40 may fail to equal or exceed the patient's inspiratory flow.[10,18] However, increasing the oxygen flow to the inlet of the nebulizer may produce a higher delivered total flow.

7.6 Hoods:

7.6.1 O_2 concentrations may vary within the hood.[10,13,18] O_2 concentrations should be measured as near the nose and mouth as possible.[11] Opening any enclosure decreases the O_2 concentration. For infants and children confined to hoods, nasal O_2 may need to be supplied during feeding and nursing care. Flows > 7 L/min are required to wash out CO_2.[13,14,31]

7.6.2 Devices can be confining and isolating.[13]

7.6.3 Concentration in a hood can be varied from 0.21 to 1.0.

7.6.4 Temperature of the gases in the hood should be maintained to provide a neutral thermal environment.[10,18,53]

7.6.5 High gas flows may produce harmful noise levels.[47,69]

7.7 Tracheostomy oxygen adapters provide

variable F_{IO_2}s. HMEs should have minimum dead-space volume especially when used with neonates.[55,70,71] Resistance within an HME can increase when water is absorbed by the hygroscopic inserts[72] or when secretions are coughed into the device.[73]

NPODD 8.0 ASSESSMENT OF NEED:

Need is determined by measurement of inadequate oxygen tensions and saturations by invasive or noninvasive methods[74,75] and/or the presence of clinical indicators as previously described.[18] Supplemental oxygen flow should be titrated to maintain adequate oxygen saturation as indicated by pulse oximetry S_{pO_2} or appropriate arterial or venous blood gas values.[76]

8.1 Nasal cannulas, nasopharyngeal catheters, and transtracheal catheters are used when the need exists to:

8.1.1 provide low-level supplemental oxygen to the infant or child;[57]

8.1.2 feed the infant without interrupting oxygen delivery;[6,77,78]

8.1.3 increase mobility.[6,77,78]

8.2 Simple oxygen masks are used to provide supplemental O_2 in the moderate range (0.35-0.50, depending on size and minute ventilation) for short periods of time (eg, during procedures, for transport, in emergency situations).[14,18,79]

8.3 Partial rebreathing masks are used to conserve the oxygen supply when higher concentrations ($F_{IO_2} > 0.4, < 0.6$) are warranted (eg, during transport).[14,18,62,79]

8.4 Non-rebreathing masks are used to deliver concentrations ≥ 0.60 or specific concentrations (as from a blender).[1,18,79]

8.5 Air-entrainment masks provide a flow of gas of predetermined precise oxygen concentration (24-40%)[12,57,66-68] that exceeds the patient's inspiratory flow. At the 50% setting, the total flow from the device may not meet the inspiratory flow.[18,79]

8.6 Air-entrainment nebulizers, although not recommended, can be used when high levels of humidity or aerosol are desired (as with a bypassed upper airway).[51,80] The patient application device can be a tracheostomy collar, face tent, aerosol mask, or blow-by arrangement.[13,18,68]

8.7 Hoods are used to provide

8.7.1 controlled F_{IO_2} in infant and small children;[10]

8.7.2 controlled F_{IO_2} and/or increased heated humidity to patients who cannot tolerate other devices;

8.7.3 controlled F_{IO_2} when the chest, abdomen, and extremities must be accessible to caregivers;[10]

8.7.4 the oxygen concentrations necessary for oxygen challenge (hyperoxia) tests in the spontaneously breathing neonate.

8.8 Tracheostomy oxygen adapters, which may or may not be coupled with HMEs, are used to deliver oxygen to a tracheostomy.[55]

NPODD 9.0 ASSESSMENT OF OUTCOME:

Outcome is assessed by determining whether the device selected produces an appropriate increase in oxygen saturation, proves to be appropriate for the patient, allows adequate patient monitoring, and facilitates patient care.

NPODD 10.0 RESOURCES:

10.1 Equipment

10.1.1 Oxygen source:

10.1.1.1 Cylinder—must meet Department of Transportation (DOT) standards, Compressed Gas Association (CGA) standards, and National Fire Protection Association (NFPA) recommendations, and appropriate regulator and wrenches must be supplied;

10.1.1.2 Concentrators (or enrichers).[81,82]

10.1.1.3 Bulk supplies should meet NFPA standards.

10.1.2 Delivery accessory equipment:

10.1.2.1 oxygen tubing;

10.1.2.2 corrugated aerosol tubing and water trap.

10.1.3 Humidifiers—No subjective or objective evidence supports routine humidification of O_2 at flows ≤ 4 L/min.[81] However, it is not known whether the use of a bubble humidifier with a nasal cannula in the neonate has benefit, and the use of a bubble humidifier can verify oxygen delivery at flows < 1 L/min. HMEs with low dead space are appropriate for short-term use in patients with artificial airways.

10.1.4 Blenders—Although blenders have been used in weaning neonates with a nasal cannula from oxygen, it appears that using a very-low flowmeter (0-200 mL) may be more reliable.[59,83]

10.1.5 Compensated, low-range flowmeters[84] adjustable in increments < 0.125 L/min.[5,58,85]

10.1.6 Oxygen analyzers—There are four principal types of oxygen analyzers; polargraphic, galvanic cell, paramagnetic, and wheatstone bridge. The polargraphic and galvanic cell are the two most commonly used and operate on an electrochemical principle.[86]

10.1.7 Noninvasive oxygen monitors—transcutaneous (TcO_2) monitor[74,86] or pulse oximeter.[75,87]

10.1.8 Nebulizer solutions—sterile water or sterile normal saline solution.

10.2 Personnel:

10.2.1 Health care providers responsible for delivery of oxygen should have demonstrated and documented knowledge and skills related to:

10.2.1.1 oxygen delivery systems and their limitations;

10.2.1.2 assembly, care, and use of oxygen delivery systems;

10.2.1.3 performance of the necessary subjective and objective assessments in order to determine effectiveness of oxygen therapy;

10.2.1.4 clinical assessment skills to recommend changes in oxygen therapy;

10.2.1.5 provision of comprehensive patient and lay caregiver instruction.

10.2.2 When supplemental oxygen is to be used out of the hospital setting, the patient and/or family member or lay caregiver should:

10.2.2.1 demonstrate proper use and understanding of oxygen delivery device;

10.2.2.2 demonstrate proper assembly, care, and cleaning of oxygen delivery device;

10.2.2.3 demonstrate an understanding of how, when, and what to report to a physician or surrogate.

NPODD 11.0 MONITORING:

11.1 Patient:

11.1.1 clinical assessment including but not limited to cardiac, pulmonary, and neurologic status and apparent work of breathing;

11.1.2 assessment of physiologic variables: noninvasive or invasive measurement of oxygen tensions or saturation in any patient treated with oxygen—within 1 hour of initiation for the neonate.[18,25,88]

11.2 Equipment:

11.2.1 All oxygen delivery systems should be checked at least once each day.[18] More frequent checks by calibrated analyzer are necessary in systems:

11.2.1.1 susceptible to variation in oxygen concentration;[18]

11.2.1.2 applied to patients with artificial airways;[18]

11.2.1.3 Continuous analysis is recommended in hoods.[89]

11.2.1.4 Oxygen should be analyzed as close as possible to the infant's face.

11.2.2 All heated delivery systems should be continuously monitored for temperature.

NPODD 12.0 FREQUENCY:

12.1 Selection of a device is made at the initiation of therapy, after careful assessment of need and patient characteristics.

12.2 The change from one type of device to another is based on a change in the patient's condition, patient preference, or ability to use a specific device. (Oxygen therapy should be administered continuously unless the need has been shown to be associated only with specific situations, eg, exercise, feeding, or other stress.)[90,91]

NPODD 13.0 INFECTION CONTROL:

13.1 Universal Precautions and measures to limit the transmission of tuberculosis must be adhered to at all times.[92,93]

13.2 Low-flow systems

13.2.1 Under normal circumstances, low-flow oxygen systems do not present clinically important risk of infection and do

not require routine replacement on the same patient.[18,94-96]

13.2.2 Nasopharyngeal catheters should be changed every 24 hours.[6]

13.2.3 Transtracheal catheters should be changed every 3 months.[97]

13.3 Reservoir systems—Under normal circumstances, reservoir systems as defined for this guideline do not present clinically important risk of infection and do not require routine replacement on the same patient.

13.4 High-flow systems

13.4.1 Large-volume nebulizers should be changed every 24 hours when applied to patients with an artificial airway.[96]

13.4.2 In the absence of definitive studies to support change-out intervals on nonintubated patients, results of institution-specific and patient-specific surveillance measures should dictate the frequency with which such equipment is replaced.

13.5 Enclosure systems—There is no recommendation regarding the frequency of changing oxyhood and reservoirs while in use on the same patient.[96]

13.6 Other devices—Between patients, subject equipment (eg, probes, oxygen sensors) to high level disinfection.[96]

13.7 Nebulizer solutions—Use only sterile fluids and dispense them aseptically.[96]

Note: It is the expert opinion of the Clinical Practice Guideline Steering Committee (2002) that some devices that are applicable to the pediatric population are not appropriate for the neonatal population.

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AARC Clinical Practice Guideline

Oxygen Therapy for Adults in the Acute Care Facility — 2002 Revision & Update

OT-AC 1.0 PROCEDURE:
The procedure addressed is the administration of oxygen therapy in the acute care facility other than with mechanical ventilators and hyperbaric chambers.

OT-AC 2.0 DEFINITION/DESCRIPTION:
Oxygen therapy is the administration of oxygen at concentrations greater than that in ambient air with the intent of treating or preventing the symptoms and manifestations of hypoxia.[1]

OT-AC 3.0 SETTING:
This Guideline is confined to oxygen administration in the acute care facility.

OT-AC 4.0 INDICATIONS:

4.1 Documented hypoxemia. Defined as a decreased P_{aO_2} in the blood below normal range.[2] P_{aO_2} of < 60 torr or S_{aO_2} of < 90% in subjects breathing room air or with P_{aO_2} and/or S_{aO_2} below desirable range for specific clinical situation.[1]

4.2 An acute care situation in which hypoxemia is suspected[1,3-6] substantiation of hypoxemia is required within an appropriate period of time following initiation of therapy.

4.3 Severe trauma[5,6]

4.4 Acute myocardial infarction[1,7]

4.5 Short-term therapy or surgical intervention (eg, post-anesthesia recovery[5,8], hip surgery[9,10])

OT-AC 5.0 CONTRAINDICATIONS:
No specific contraindications to oxygen therapy exist when indications are judged to be present.

OT-AC 6.0 PRECAUTIONS AND/OR POSSIBLE COMPLICATIONS:

6.1 With $P_{aO_2} \geq 60$ torr, ventilatory depression may occur in spontaneously breathing patients with elevated P_{aCO_2}.[6,11-14]

6.2 With $F_{IO_2} \geq 0.5$, absorption atelectasis, oxygen toxicity, and/or depression of ciliary and/or leukocytic function may occur.[12,15,16]

6.3 Supplemental oxygen should be administered with caution to patients suffering from paraquat poisoning[17] and to patients receiving bleomycin.[18]

6.4 During laser bronchoscopy, minimal levels of supplemental oxygen should be used to avoid intratracheal ignition.[19]

6.5 Fire hazard is increased in the presence of increased oxygen concentrations.

6.6 Bacterial contamination associated with certain nebulization and humidification systems is a possible hazard.[20-22]

OT-AC 7.0 LIMITATIONS OF PROCEDURE:
Oxygen therapy has only limited benefit for the treatment of hypoxia due to anemia, and benefit may be limited with circulatory disturbances. Oxygen therapy should not be used in lieu of but in addition to mechanical ventilation when ventilatory support is indicated.

OT-AC 8.0 ASSESSMENT OF NEED:
Need is determined by measurement of inadequate oxygen tensions and/or saturations, by invasive or noninvasive methods, and/or the presence of clinical indicators as previously described.

OT-AC 9.0 ASSESSMENT OF OUTCOME:
Outcome is determined by clinical and physiologic assessment to establish adequacy of patient response to therapy.

OT-AC 10.0 RESOURCES:
For other types of oxygen delivery devices used outside of the acute care facility, reference the AARC Clinical Practice Guideline: Oxygen Therapy in the Home or Extended Care Facility for further description. Respir Care 1992;37(8):918-922.

10.1 Equipment

10.1.1 Low-flow systems deliver 100% (ie, $F_{DO_2} = 1.0$) oxygen at flows that are less than the patient's inspiratory flowrate (ie, the delivered oxygen is diluted with room air) and, thus, the oxygen concentration inhaled (F_{IO_2}) may be low or high, depending on the specific device and the patient's inspiratory flowrate.[23,24]

10.1.1.1 Nasal cannulas can provide 24-40% oxygen with flowrates up to 6 L/min in adults (depending on ventilatory pattern).[1] Oxygen supplied via nasal cannula at flowrates ≤ 4 L/min need not be humidified.[25,26] Care must be taken when assigning an estimated F_{IO_2} to patients as this low-flow system can have great fluctuations.[27]

10.1.1.2 Simple oxygen masks can provide 35-50% F_{IO_2}, depending on fit, at flowrates from 5-10 L/min. Flowrates should be maintained at 5 L/min or more in order to avoid rebreathing exhaled CO_2 that can be retained in the mask.[1,20,28] Caution should be taken when using a simple mask where accurate delivery of low concentrations of oxygen is required.[29] Long-term use of simple mask can lead to skin irritation and pressure sores.[30]

10.1.1.3 Partial rebreathing mask is a simple mask with a reservoir bag. Oxygen flow should always be supplied to maintain the reservior bag at least one third to one half full on inspiration. At a flow of 6-10 L/min the system can provide 40-70% oxygen. It is considered a low-flow system. The non-rebreathing mask is similar to the partial rebreathing mask except it has a series of one-way valves. One valve is placed between the bag and the mask to prevent exhaled air from returning to the bag. There should be a minimum flow of 10 L/min. The delivered F_{IO_2} of this system is 60-80%.

10.1.1.4 Patients who have been receiving transtracheal oxygen at home may continue to receive oxygen by this method in the acute care facility setting provided no problems present. If difficulties related to the transtracheal route of administration appear, oxygenation should be assured by other means.

10.1.2 High-flow systems deliver a prescribed gas mixture—either high or low F_{DO_2} at flowrates that exceed patient demand.[23,24,31]

10.1.2.1 Currently available air-entrainment masks can accurately deliver predetermined oxygen concentration to the trachea up to 40%. Jet-mixing masks rated at 35% or higher usually do not deliver flowrates adequate to meet the inspiratory flowrates of adults in respiratory distress.[7,24,31,32]

10.1.2.2 Aerosol masks, tracheostomy collars, T-tube adapters, and face tents can be used with high-flow supplemental oxygen systems. A continuous aerosol generator or large-volume reservoir humidifier can humidify the gas flow. Some aerosol generators cannot provide adequate flows at high oxygen concentrations.[1]

10.2 Personnel

10.2.1 Level I personnel—ie, any person who has adequately demonstrated the ability to perform the task—may check and document that a device is being used appropriately and the flow is as prescribed.

10.2.2 Level II personnel—licensed or credentialed respiratory care practitioners or persons with equivalent training and documented ability to perform the tasks—may assess patients, initiate and monitor oxygen delivery systems, and recommend changes in therapy.

OT-AC 11.0 MONITORING:

11.1 Patient

11.1.1 clinical assessment including but not limited to cardiac, pulmonary, and neurologic status

11.1.2 assessment of physiologic parameters: measurement of P_{aO_2}s or saturation in any patient treated with oxygen. An appropriate oxygen therapy utilization protocol is suggested as a method to decrease

waste and to realize increased cost savings.[33] Consider need/indication to adjust F_{IO_2} for increased levels of activity and exercise.

11.1.2.1 in conjunction with the initiation of therapy; or

11.1.2.2 within 12 hours of initiation with $F_{IO_2} < 0.40$

11.1.2.3 within 8 hours, with $F_{IO_2} \geq 0.40$ (including postanesthesia recovery)

11.1.2.4 within 72 hours in acute myocardial infarction[7]

11.1.2.5 within 2 hours for any patient with the principal diagnosis of COPD

11.2 Equipment

11.2.1 All oxygen delivery systems should be checked at least once per day.

11.2.2 More frequent checks by calibrated analyzer are necessary in systems

11.2.2.1 susceptible to variation in oxygen concentration (eg, high-flow blending systems)

11.2.2.2 applied to patients with artificial airways

11.2.2.3 delivering a heated gas mixture

11.2.2.4 applied to patients who are clinically unstable or who require an F_{IO_2} of 0.50 or higher.

11.2.3 Care should be taken to avoid interruption of oxygen therapy in situations including ambulation or transport for procedures.

OT-AC 12.0 FREQUENCY:

Oxygen therapy should be administered continuously unless the need has been shown to be associated only with specific situations (eg, exercise and sleep).

OT-AC 13.0 INFECTION CONTROL:

Under normal circumstances, low-flow oxygen systems (including cannulas and simple masks) do not present clinically important risk of infection and need not be routinely replaced.[1] High-flow systems that employ heated humidifiers and aerosol generators, particularly when applied to patients with artificial airways, can pose important risk of infection. In the absence of definitive studies to support change-out intervals, results of institution-specific and patient-specific surveillance measures should dictate the frequency with which such equipment is replaced.

Revised by Thomas J Kallstrom RRT FAARC, Fairview Hospital, Cleveland, OH, and approved by the 2002 CPG Steering Committee.

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AARC Clinical Practice Guideline

In-Hospital Transport of the Mechanically Ventilated Patient — 2002 Revision & Update

TMV 1.0 PROCEDURE:
Transportation of a mechanically ventilated patient for diagnostic or therapeutic procedures.

TMV 2.0 DESCRIPTION/DEFINITION:
Transportation of mechanically ventilated patients for diagnostic or therapeutic procedures is always associated with a degree of risk.[1-9] Every attempt should be made to assure that monitoring, ventilation, oxygenation, and patient care remain constant during movement. Patient transport includes preparation, movement to and from, and time spent at destination.

TMV 3.0 SETTINGS:
This guideline is intended for the critical care and acute care inpatient setting.

TMV 4.0 INDICATIONS:
Transportation of mechanically ventilated patients should only be undertaken following a careful evaluation of the risk-benefit ratio.

TMV 5.0 CONTRAINDICATIONS:
Transportation of the mechanically ventilated patient should not be undertaken until a complete analysis of potential risks and benefits has been accomplished.

5.1 Contraindications include

5.1.1 inability to provide adequate oxygenation and ventilation during transport either by manual ventilation, portable ventilator, or standard intensive care unit ventilator,[1,10-12]

5.1.2 inability to maintain acceptable hemodynamic performance during transport,[13-15]

5.1.3 inability to adequately monitor patient cardiopulmonary status during transport,[14]

5.1.4 inability to maintain airway control during transport.

5.1.5 Transport should not be undertaken unless all the necessary members of the transport team are present.

TMV 6.0 HAZARDS & COMPLICATIONS:
Hazards and complications of transport include the following:

6.1 Hyperventilation during manual ventilation may cause respiratory alkalosis, cardiac dysrhythmias, and hypotension.[1,10,11]

6.2 Loss of PEEP/CPAP may result in hypoxemia or shock.[16,17]

6.3 Position changes may result in hypotension, hypercarbia, and hypoxemia.[14]

6.4 Tachycardia and other dysrhythmias have been associated with transport.[13-15]

6.5 Equipment failure can result in inaccurate data or loss of monitoring capabilities.[8,9]

6.6 Inadvertent disconnection of intravenous access for pharmacologic agents may result in hemodynamic instability.[8,9,13]

6.7 Movement may cause disconnection from ventilatory support and respiratory compromise.[16,18]

6.8 Movement may result in accidental extubation.[13,19]

6.9 Movement may result in accidental removal of vascular access.[3-5,8,9]

6.10 Loss of oxygen supply may lead to hypoxemia.

6.11 Ventilator-associated pneumonia has been associated with transport.[20]

TMV 7.0 LIMITATIONS OF METHOD:
The literature suggests that nearly two thirds of all transports for diagnostic studies fail to yield results that affect patient care.[8,9]

TMV 8.0 ASSESSMENT OF NEED:
The necessity and safety for transport should be as-

sessed by the multidisciplinary team of health care providers, eg, respiratory therapist, physician, nurse. The risks of transport should be weighed against the potential benefits from the diagnostic or therapeutic procedure to be performed.

TMV 9.0 ASSESSMENT OF OUTCOME:
The safe arrival of the mechanically ventilated patient at his/ her destination is the indicator of a favorable outcome.

TMV 10.0 RESOURCES:

10.1 Equipment

10.1.1 Emergency airway management supplies should be available and checked for operation before transport

10.1.2 Portable oxygen source of adequate volume

10.1.3 A self-inflating bag and mask of appropriate size

10.1.4 Transport ventilators have been shown to provide more constant ventilation than manual ventilation in some instances. If a transport ventilator is used, it should:[1,10-12,21,22]

10.1.4.1 have sufficient portable power supply for the duration of transport;[23]

10.1.4.2 have independent control of tidal volume and respiratory frequency;[16]

10.1.4.3 be able to provide full ventilatory support as in assist-control or intermittent mechanical ventilation (not necessarily both);

10.1.4.4 deliver a constant volume in the face of changing pulmonary impedance;

10.1.4.5 monitor airway pressure;

10.1.4.6 provide a disconnect alarm;

10.1.4.7 be capable of providing PEEP;

10.1.4.8 provide an F_{IO_2} of 1.0.

10.1.5 A pulse oximeter is desirable.

10.1.6 Appropriate pharmacologic agents should be readily available.

10.1.7 Portable monitor should display ECG and heart rate and provide at least one channel for vascular pressure measurement.

10.1.8 An appropriate hygroscopic condenser humidifier should be used to provide humidification during transport.

10.1.9 Stethoscope

10.1.10 Hand-held spirometer for tidal volume measurement

10.2 Personnel: All mechanically ventilated patients should be accompanied by a registered nurse and a respiratory therapist during the entire transport.

10.2.1 At least one team member must be proficient in managing the airway in the event of accidental extubation.

10.2.2 At least one team member should be proficient in operating and troubleshooting all of the equipment described in Section 10.1.

TMV 11.0 MONITORING:
Monitoring provided during transport should be similar to that during stationary care.

11.1 Electrocardiograph should be continuously monitored for heart rate and dysrhythmias.

11.2 Blood pressure should be monitored continuously if invasive lines are present. In the absence of invasive monitoring, blood pressure should be measured intermittently via sphygmomanometer.

11.3 Respiratory rate should be monitored intermittently.

11.4 Airway pressures should be monitored if a transport ventilator is used.[24]

11.5 Tidal volume should be monitored intermittently to assure appropriate ventilation.[25]

11.6 Continuous pulse oximetry is appropriate during transport of all mechanically ventilated patients.

11.7 Breath sounds should be monitored intermittently.

TMV 12.0 FREQUENCY:
Patients should be transported only when indications are present as described in Section 4.

TMV 13.0 INFECTION CONTROL:

13.1 Universal Precautions should be observed.[26]

13.2 All equipment should be disinfected between patients.

13.3 Centers for Disease Control and Prevention recommendations for control of exposure to tuberculosis and droplet nuclei are to be im-

plemented when patient is known or suspected to be immunosuppressed, is known to have tuberculosis, or has other risk factors for the disease.[27]

Revised by David W Chang EdD RRT, Athens Technical College, Athens GA, and approved by the 2002 CPG Steering Committee.

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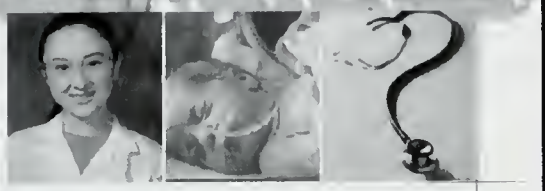

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SATURDAY, Dec. 1

- ❑ 1. Keynote Address, D. Satcher
- ❑ 5. Tracking Flow of Residents, D.N. Muse
- ❑ 6. (6.) Role of Educators, E.A. Becker &
  (7.) Role of Managers, K.J. Stewart &
  (8.) Panel Discussion, M.Traband
- ❑ 9. Errors, Law and RCPs, A.L. DeWitt
- ❑ 10. Human Error: Changing Our Perceptions, J.W. Salyer
- ❑ 11. Patient Abuse in the Home: What Is It and What Are Your Responsibilities? D. Roberts
- ❑ 12. Assessing the Right Competencies for Home Care Therapists, S.L. Moreau
- ❑ 13. Discharging the Complex Pediatric Patient to the Home, J.P. Stegmaier
- ❑ 14. Does Economics Drive Home Care? R.W. McCoy
- ❑ 15. Surfactant Therapy, R.J. Rodriguez
- ❑ 16. Neonatal Update: Inhaled Nitric Oxide in the Neonate, S.R. Seidner
- ❑ 17. Mgt. Strategies for Prevention of BPD, R.J. Rodriguez
- ❑ 18. Overview of Dying and End-of-Life Care in America, P.A. Selecky
- ❑ 19. Withholding and Withdrawing Ventilation, R. Acevedo
- ❑ 20. Role of the Respiratory Therapist in Palliative Care, H.M. Sorenson
- ❑ 21. (21.) Building Coalitions, J.B. Fink
  (22.) Reaching Patients and Physicians with "Baylor's Rules of Two"!, G.E. Lawrence
- ❑ 23. (23.) Regional Asthma Center, L. Parish-Cooper
  (24.) Promoting Public Awareness: Successful Strategies, S. Blonshine
- ❑ 25. (25.) Sleep Disordered Breathing, M.B. Dunning III
  (26.) Protocols for Standardized Sleep Testing, R.N. Turner
- ❑ 27. (27.) New Polysomnograph Technologist Training Guidelines, R.N. Turner
  (28.) Survival Techniques for Shift Workers, M.B. Dunning
- ❑ 31 A. Issues & Trends in Allied Health Leadership, M. Harrington, Developing Your Career for Maximum Professional Effectiveness, M. W. Runge
  Breakout #1: Leadership in Clinical Practice, G. Gaebler
  Breakout #3: Leadership in Management, J.D. Kimble (Needs overtime tape)
- ❑ 31 B. Developing Professional Leadership, R. Krueger
  Success Stories, J.C. Bolden , D. Johnson
- ❑ 31 C. #2: Leadership in Clinical Research, A.B. Adams &
  #4: Leadership in Education, L. Van Scoder
- ❑ 34. Humor at the Bedside, M. Graves

SUNDAY, Dec. 2

- ❑ 35. Egan Lecture — Chronic Obstructive Pulmonary Disease - On the Exponential Curve of Progress, J.E. Heffner
- ❑ 36. Pulmonary Rehabilitation, B.W. Carlin
- ❑ 37. High Frequency Oscillatory Ventilation for ARDS: State of the Art 2001, S. Derdak
- ❑ 38. "The Ins and Outs" of Lung Transplantation, S.M. Levine MD, C.G. Durbin Jr.
- ❑ 39. Perspectives from Respiratory Care and Nursing, P. Carroll, & A.L. DeWitt Jr.
- ❑ 40. Risk Management and Respiratory Care, S.J. Price
- ❑ 41. Competency: Is Your Staff Ready? J.R. Edens
- ❑ 42. Managing Protocols Based on CPGs, E. Hoisington
- ❑ 45. A New Opportunity, C.M. Kercsmar
- ❑ 46. Questions for the Attorney, A.L. DeWitt
- ❑ 47. JCAHO: Showcasing Your Best, R.M. Ford
- ❑ 48. Justifying Human Resources: Best Use of a Valuable Asset, R.M. Ford
- ❑ 49. (49.) It's All Black and White, B. Yawn &
  (50.) Managing to Breathe via Internet, C. Olson
- ❑ 51. The Dirty Dozen, N. Sander
- ❑ 52. Selecting Clinical and Functional Outcomes in LTOT, T.L. Petty
- ❑ 53. Selecting Fiscal and Satisfaction Outcomes in LTOT, T.W. B
- ❑ 54. Measuring and Collecting Outcome Data, D.C. Shelledy
- ❑ 55. Using LTOT Outcome Data, R. Fary
- ❑ 56. Outcome Data Panel Discussion, T.W. Buckley, R. Fary, T.I Petty, and D.C. Shelledy
- ❑ 57. Smoking Cessation, D.D. Gardner
- ❑ 58. New Strategies for Treating Tobacco Use and Dependence, Cohn
- ❑ 59. How Respiratory Care Managers Can Implement Tobacco Education Programs for Community-Wide Effect, S.M. Ciarl
- ❑ 60. Secondhand Smoke — What Can Health Care Professionals About It? A. French, B. Huffman
- ❑ 61. (61.) Evolution and Rationale of the Low VT Protocol, R. Br
  (62.) Practical Aspects of Implementing the Low VT Protoc Kallet
- ❑ 63. (63.) Can the ARDS Network Protocol Be Modified?, N.R. MacIntyre &
  (64.) How and Why to Use Recruitment Maneuvers in Low Ventilation, N.R. MacIntyre
- ❑ 65. (65.) Normal Mechanisms of Airway Clearance and Proble from the ICU to Home, B.K. Rubin &
  (66.) Positioning vs. Postural Drainage, J. Fink &
  (67.) Breathing Maneuvers, M. MacIllwaine
- ❑ 68. (68.) Positive Pressure Techniques for Airway Clearance, M Mahlmeister &
  (69.) High Frequency Oscillation of the Airway and Chest V J. Fink &
  (70.) Airway Clearance and the Artificial Airways, R. Lewi
- ❑ 71. (71.) Pharmaceutic Approach to Airway Clearance, B.K. Rub
  (72.) Strategies for the Pediatric Patient, K.L. Davidson &
  (73.) Airway Clearance Strategies for the Vent Patient at Ho C. Lapin
- ❑ 74. RSV: Management and Treatment Advances, C.M. Kercsma
- ❑ 75. Test Your Skills on the State of the Profession, M.F. Traban
- ❑ 76. How to Survive as a Multi-Department Manager, S.J. Price
- ❑ 79. Technological Advances in Noninvasive Respiratory Monito G. Devine
- ❑ 80. Monitoring on the General Floor: What's the Future Going Like? P.T. Sharkey
- ❑ 81. A Proposal for Evidence-Based Practice in Neonatal Resuscitation, W.D. Rich
- ❑ 82. Classifying Asthma Patients through Symptom Analysis, T. Kallstrom
- ❑ 83. How Not to Run a Respiratory Care Service, K.L. Shrake

MONDAY, Dec. 3

- ❑ 84. Kittredge Lecture — Mechanical Ventilation: How Did We Here and Where Are We Going?, R.D. Branson
- ❑ 85. Intrapulmonary Percussive Ventilation, C. Diaz
- ❑ 88. Exercise Training: Are You Training Your Patients at the Ri Exercise Target? B.W. Carlin
- ❑ 89. Water Exercise in Pulmonary Rehabilitation Programs, M.W. M
- ❑ 90. The Impact of the Institute of Medicine's Report for Respira Care Providers, S.P. Giordano
- ❑ 91. Evidence-Based Respiratory Care, D.J. Pierson
- ❑ 92. Managing Chronic Medical Conditions in the New Care De Framework, P.J. Dunne
- ❑ 93. (93.) Applying Case Management Principles in Home Care, Stegmaier &
  (94.) Developing a Cost Model For Delivery of LTOT, T.W. Bi
- ❑ 95. (95.) What I Wish All Home Care Therapists Knew, B. Roger
  (96.) LTOT Utilization: The Bigger Picture! G. Spratt
- ❑ 97. (97.) Modifying Treatment Plans For the Aging Lung, T.S. LeGrand &
  (98.) Seeing Health Care through the Eyes of the Elderly, H Sorenson

❑ 99. (99.) Caring for the Older Pneumonia Patient, T.S. LeGrand &
(100.) Senior Pulmonary Assessment, H.M. Sorenson
❑ 101. Interpretation of PFTs by RCPs, J.O. Nilsestuen
❑ 102. Results of a National Study, J.M. Boyle
❑ 103. Agency Updates ñ Through the eyes of the AARC, ARCF, CoARC and NBRC, C.P. Wiezalis, M.T. Amato, M. Welch Jr, and J. Long-Goding
❑ 104. Munchausen by Proxy, D. Roberts
❑ 105. Opening the Black Box during Mechanical Ventilation, M. Amato
❑ 107. (107.) Documenting Respiratory Practice Beyond the USA, J. Long-Goding H.L. Garza &
(108.) Comparison of Two Methods for Administration of Inhaled Salbutamol, H.L. Molina
❑ 109. (109.) The Brazilian Experience, L.G. Ghion &
(110.) The United Kingdom Experience, A. Hynes
❑ 111. (111.) The Italian Experience, S. Thompson &
(112.) The Japanese Experience, H. Etchuya
❑ 113. (113.)The North America Experience, A. King &
(114.) The Personal Experience — A Patient's View, B. Rogers, & Panel Discussion
❑ 115. Creating an Effective Multi-Cultural Work Environment, K.L. Shrake
❑ 116. Bronchiolitis Care Path, J.W. Salyer
❑ 117. Pediatric Asthma: Success with Therapist-Driven Protocols, T.R. Myers
❑ 118. Developing a Pediatric Consult Program, J.W. Salyer
❑ 119. Lung Protective Strategies for Status Asthmaticus, M.K. Brown
❑ 120. Acute Care of the Adult Asthmatic, J.J. Peters
❑ 121. Bronchodilator Resuscitation in the ER, J. Fink
❑ 122. Case Presentations of Pulmonary X-Rays, C.G. Durbin
❑ 123. (123.) P/V Curves Should Be Routinely Used in Ventilatory Management of ALI, K. Hargett, R.M. Kacmarek &
(124.) Heliox is the Front-Line Treatment for Management of Acute Asthma, T.R. Myer, C.M. Keresmar
❑ 125. New Modes of Mechanical Ventilation Are Necessary for Management of Patients with ARF, N.R. MacIntyre, R.D. Branson
❑ 126. Oxygen Toxicity in the ICU Is a Myth, D.J. Pierson, C.G. Durbin Jr
❑ 131. An Answer to Respiratory Care Re-Engineering, D.C. Oberly
❑ 132. What's Right and What's Wrong with Cardio-Pulmonary Exercise Tests, M.W. Millard
❑ 133. The Amazing History of Pulmonary Medicine, G.W. Lantz
❑ 134. New Uses for an Old Therapy, M.K. Brown

TUESDAY, Dec. 4

❑ 135. Which One for ARDS? M. Amato
❑ 136. What It Is and How You Can Help, R.R. Fluck
❑ 137. Prospective Payment System (PPS)
❑ 138. Models of Success for RCPs, K.A. Cornish
❑ 139. Wave Form Analysis, J.O. Nilsestuen
❑ 140. Diagnostic Testing: Pre- and Post-Lung Transplant, C.M. Foss
❑ 141. Lung Transplantation from the Patient's Perspective, M. Pierce
❑ 142. Apples to Apples: First Line Therapy, S. Jenkinson
❑ 143. Safety of First Line Options, T.A. Mahr,
❑ 144. Advantages of Combination Therapy, J. Bloom
❑ 145. Advances in Delivery: Does Particle Size Matter? T.A. Mahr
❑ 146. NPPV Without Quantifiable Evidence: The Legal and Medic Ramifications, P.K. Blakely
❑ 147. Using Protocols in Respiratory Home Care, G. Spratt
❑ 148. Telemedicine and the Home Care Respiratory Therapist, P.K Blakely
❑ 149. Corporate Compliance Plan: What the OIG Is Really Saying Dunne
❑ 150. (150.) How To Come Up with a Good Research Question, C Durbin Jr &
(151.) How to Get a Research Project Done in Your Departn J. Ward
❑ 152. (152.) How to Write an Open Forum Abstract That Will Be Accepted, D.J. Pierson &
(153.) How to Make an Effective Poster for Presentation at t Open Forum, D.C. Shelledy &
(154.) How to Prepare and Deliver an Effective Oral, Summ of Your Project, R.S. Campbell
❑ 155. Pulmonary Hypertension and the Respiratory Therapist, R.L Rosenblatt
❑ 156. Management of Amyotrophic Lateral Sclerosis, C.E. Jackson
❑ 157. Insight to the Profession, Panel
❑ 158. Weaning from Mechanical Ventilation, N.R. MacIntyre
❑ 159. Treatment of the Adult Patient with Cystic Fibrosis, R.L. Rosenblatt
❑ 160. Recent Developments in the Management of Meconium Aspiration, R. Castro
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American Association for Respiratory Care

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An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC.

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Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field

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Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

Please read the eligibility requirements for each of the classifications above, then complete the form. All information requested must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
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- ☐ Supervisor
- ☐ Diagnostic Technologist
- ☐ Instructor/Educator
- ☐ Other, specify _____
- ☐ Therapist/Technician
- ☐ Medical Director
- ☐ Student
- ☐ Staff Nurse

Type of Business

- ☐ Educational Institution
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Hospital/Acute Care
- ☐ Other, specify _____
- ☐ Manufacturer or supplier
- ☐ Outpatient Clinic
- ☐ Physician office
- ☐ Skilled Nursing Facility

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
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American Association for Respiratory Care **MEMBERSHIP APPLICATION**

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Payment must accompany your application to the AARC. Fees are for 12 months. These fees contain the $12.50 new members processing fee. Renewing members (except students) can deduct $12.50.

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Article in journal supplement: (Journals differ in numbering and identifying supplements. Supply information sufficient to allow retrieval.)
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Editorial with no author given:
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Letter in journal:
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Cairo JM, Pilbeam SP. Mosby's respiratory care equipment, 6th ed. St Louis: Mosby; 1999:76-85.

Corporate author book:
American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Chapter in book with editor(s):
Isono S. Upper airway muscle function during sleep. In: Loughlin GM, Carroll JL, Marcus CL, editors. Sleep and breathing in children: a developmental approach. (Lung Biology in Health and Disease, Vol 147, Claude Lenfant, Executive Editor.) New York/Basel: Marcel Dekker; 2000:261-291.

World Wide Web
American Lung Association. Trends in pneumonia, influenza, and acute respiratory conditions mortality and morbidity. February, 2000. http://www.lungusa.org/data. Accessed November 20, 2000.

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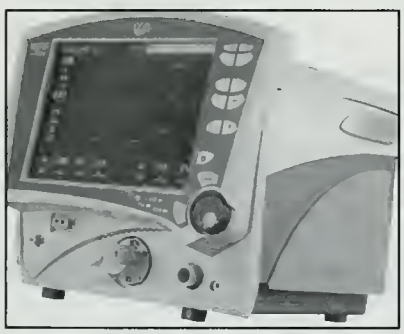

Ventilator. VIASYS Healthcare has announced that it has received FDA approval of the Vela™ ventilator for commercial distribution in the United States. The release letter states that Vela may be "used to provide continuous or intermittent mechanical ventilatory support." According to the manufacturer, the Vela is a mid-range ventilator intended for use in both intensive care and subacute settings. Its compact size and integrated turbine supports the patient throughout the entire continuum of care: ER, surgery, ICU, and recovery, says VIASYS. According to their press release, the Vela incorporates touch screen control into an intuitive Graphical User Interface (GUI) to provide easy access to critical patient data. The LCD screen clearly displays breaths as a waveform or loop for in-depth analysis, according to the manufacturer. For more information from VIASYS Healthcare, circle number 176 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Valved Holding Chamber. PARI Respiratory Equipment Inc has recently introduced its newest aerosol delivery product for the North American respiratory market—the PARI Vortex™, a non-electrostatic valved holding chamber. According to PARI, the Vortex is made of metal, allowing patients a more consistent delivery of their inhaled medications when using a metered-dose inhaler because, unlike plastic holding chambers, the Vortex does not maintain an electrostatic charge. This means that more of the medication can reach the lungs, says the manufacturer. Another feature is the cyclonic inspiratory flow pattern which, according to PARI, further enhances aerosol delivery for patients with low tidal volumes, like those of young children and infants. For more information from PARI Respiratory Equipment, circle number 177 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Breath Simulation Module. Michigan Instruments Inc introduces a Breath Simulation Module. This new module will turn existing Michigan Instruments lung models into a spontaneously breathing lung simulation system, according to Michigan Instruments. The module simulates a spontaneously breathing patient for training or studying the use CPAP, IMV, SIMV, pressure support, work of breathing, or other ventilator modes designed for use with breathing patients. The manufacturer further says that this device can also test/troubleshoot products designed to support a spontaneously breathing patient. For more information from Michigan Instruments, circle number 178 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Pulse Oximetry System. Nellcor has recently introduced the *OxiMax*™ Pulse Oximetry System, which they describe as a revolutionary technology platform that greatly expands the clinical potential of pulse oximetry monitoring. The *OxiMax* Pulse Oximetry System includes the new Nellcor N-595 Pulse Oximeter and Nellcor's new family of intelligent, interactive *OxiMax* sensors. The *OxiMax* system is used to continuously monitor arterial oxygen saturation and pulse rate for patients of all ages. Nellcor says that a communication link between the monitor and sensor empowers the N-595 Pulse Oximeter to display valuable information to caregivers. Nellcor has embedded a digital memory chip in the sensor itself that contains all the calibration and operating data for that sensor design. This gives the *OxiMax* monitor the versatility to operate more accurately with a diverse range of sensor designs, allowing pulse oximetry sensor innovations not possible in the past, according to the manufacturer. For more information from Nellcor, circle number 179 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Scheduled Professor's Rounds 2002

Withholding and Withdrawing Life Support in the ICU — Gordon D Rubenfeld MDMSc / Richard D Branson BA RRT FAARC — Videotape Available

Weaning from Mechanical Ventilation: New Insights, New Guidelines — Neil R MacIntyre MD FAARC/ Dean R Hess PhD RRT FAARC — Videotape Available

Neonatal and Pediatric Ventilators: What's the Difference? — Mark J Heulitt MD FAARC/ Richard D Branson BA RRT FAARC — Live May 21; Audio June 25

Ventilator Graphs: What's With That Wave? — Jon O Nilsestuen PhD RRT FAARC/ Richard D Branson BA RRT FAARC —Live July 16; Audio August 13

Talking with Patients and Families About Death and Dying— Helen M Sorensen MA RRT FAARC/ David J Pierson MD FAARC —Live August 20; Audio September 17

Pressure vs Volume Ventilation: Does It Matter? — Robert S Campbell RRT FAARC/ Richard D Branson BA RRT FAARC — Live September 10/ Audio October 8

Inpatient Management of COPD — Randall Rosenblatt MD/ David J Pierson MD FAARC — Live October 22; Audio November 12

High-Frequency Oscillatory Ventilation — Thomas E Stewart MD/ Richard D Branson BA RRT FAARC — Live November 19; Audio December 10

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
- Current job listings
- American Respiratory Care Foundation fellowships, grants, & awards
- Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

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Asthma Management Model System
http://www.nhlbi.nih.gov

Keys to Professional Excellence
http://www.aarc.org/keys/

Committee on Accreditation for Respiratory Care
http://www.coarc.com

The National Board for Respiratory Care— Examination Fees for 2002

Examination	Examination Fees
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CPFT	$200 (new applicant) $170 (reapplicant)
RPFT	$250 (new applicant) $220 (reapplicant)
RRT (Written & CSE)	$190 (new) $150 (reapplicant) written only $200 (new and reapplicant) CSE only $390 (new) $350 (reapplicant) both

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156, or e-mail: nbrc-info@nbrc.org

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $50 for members and $60 for nonmembers and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Calendar of Events

Date	AARC & State Society Programs	Contact
June 11–13	New Jersey and New York State Societies for Respiratory Care, 15th Annual Spring Forum; **Round Top, NY**	Ken Wyka (NJ) at (201) 725-2528; or Bob Fluck (NY) at (315) 464-5580
June 14–16	AARC Summer Forum; **Vail, CO**	AARC, (972) 243-2272, www.aarc.org
June 16	AARC/EPA Asthma and Indoor Air Environment Workshop; **Keystone, CO**	AARC, (972) 243-2272, www.aarc.org
June 16–17	AARC Asthma Disease Management Course; **Vail, CO**	AARC, (972) 243-2272, www.aarc.org
June 19–21	ISRC 34th Annual Convention & Exhibition; **Oakbrook Terrace, IL**	General information, Kelli DeBerry (847) 981-3581; vendors, Larry Dastych (847) 618-3180
June 27	Asthma Information Review course; **Grand Rapids, MI**	Michigan Society for Respiratory Care, (734) 677-6772, www.michiganrc.com
June 27–29	34th Annual California Society for Respiratory Care Convention; **Universal City, CA**	CSRC Executive Office (888) 730-CSRC or www.csrc.org
July 17–19	TSRC 31st Annual Convention and Exhibition; **San Antonio, TX**	TSRC Executive Office (972) 680-2455
July 27	Asthma Information Review course; **Flint, MI**	Michigan Society for Respiratory Care, (734) 677-6772, www.michiganrc.com
Aug. 1–3	OSRC's Contemporary Concepts in Respiratory Care; **Independence, OH**	Tom Kallstrom at tom.kallstrom@fairviewhospital.org; or Jeffrey Davis at davisj@ccf.org
Aug. 1–4	Georgia Society for Respiratory Care's Summer Meeting; **St. Simons, GA**	David Ellwanger, (770) 991-8558, ellwanger_david@promina.org
Aug. 14–16	31st TriState Respiratory Care Conference; **Biloxi, MS**	Linda Berry (205) 343-8475; www.tsrcc.org
Sept. 5–6	Vermont/New Hampshire Society for Respiratory Care Annual Meeting; **Killington, VT**	Xan Gallup (800) 333-8095, ext. 360 or xgallup@merriam-graves.com
Sept. 5–7	TSRC North Region – Pineywoods District 16th Annual Fall Seminar; **Lufkin, TX**	Pam Hollis (409) 639-7006
Sept. 11–12	MSRC 25th Annual State Conference; **Sturbridge, MA**	Frances Smith (508) 833-9893, fsmith91@hotmail.com
Sept. 12–13	PSRC Southwest District's 29th Annual Seminar in Pulmonary and Sleep Medicine; **Pittsburgh, PA**	Gail Varcelotti (724) 941-8792, SWDistrict@psrc.net
Sept. 12–13	Alabama Society for Respiratory Care Annual Meeting; **Birmingham, AL**	Bill Pruitt (251) 434-3405 or wpruitt@jaguar1.usouthal.edu
Sept. 19–20	Kansas Respiratory Care Society Western Kansas Conference; **Hays, KS**	www.krcs.org or e—mail Julia Downs at jddrrt@yahoo.com
Oct. 5–8	AARC 48th International Respiratory Congress; **Tampa, FL**	AARC, (972) 243-2272, www.aarc.org

Authors in This Issue

Austin, Paul N 667
Branson, Richard D 667
Brown, Melissa K 702
Campbell, Robert S 667
Carmichael, Tina 688
Cattapan, Steven E 693
Chandrasekhar, Arcot J 693
Crogan, Karen J 703
Dahdah, Nagib S 662
Demers, Robert R 696
Foland, Jason A 662
Granton, John T 675
Hart, Mary 700
Hatipoglu, Umur 693
Henke, Markus O 706
Hess, Dean R 696
Holgate, Stephen T 705
Johannigman, Jay A 667
Kemp, James P 704
Kesten, Steven 675
Laghi, Franco 693
Laussen, Peter C 688
Levine, Donna 688
Mammel, Mark C 682
Meyers, Patricia A 682
Mhanna, Maroun J 662
Millard, Mark W 700
Rubin, Bruce K 706
Shapiro, Colin 675
Super, Dennis M 662
Trusty, Robert 682
Venkayya, Rajeev V 701
Worwa, Catherine 682

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119	120	121	122	123	124	125	126	127	128	129	130	131	132	133	134	135	136
137	138	139	140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169	170	171	172
173	174	175	176	177	178	179	180	181	182	183	184	185	186	187	188	189	190
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Chandrasekhar, Arcot J 693
Crogan, Karen J 703
Dahdah, Nagib S 662
Demers, Robert R 696
Foland, Jason A 662
Granton, John T 675
Hart, Mary 700
Hatipoglu, Umur 693
Henke, Markus O . . .
Hess, Dean R . . . . . .
Holgate, Stephen T .
Johannigman, Jay A 667
Kemp, James P 704
Kesten, Steven 675
Laghi, Franco 693
Laussen, Peter C 688
Levine, Donna 688
Mammel, Mark C 682
Meyers, Patricia A 682
Mhanna, Maroun J 662
Millard, Mark W 700
Rubin, Bruce K 706
Shapiro, Colin 675
Super, Dennis M 662
Trusty, Robert 682

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1 McIlwaine, P.M., Wong, L.T., Peacock, D., Davidson, G.F. "Long-term comparative trial of conventional postural drainage and percussion versus positive expiratory pressure physiotherapy in the treatment of cystic fibrosis." *The Journal of Pediatrics*, October 1997.

2 Protocol of the Month, University of Pittsburgh Medical Center, Department of Respiratory Care. *AARC Times*, May 1997.

3 Mahlmeister MJ, Fink JB, Hoffman GL, Fifer LF, "Positive-expiratory-pressure mask therapy: Theoretical and Practical Considerations and a Review of the Literature", *Respiratory Care*, 1991; 36:1218-1230.

4 "AARC Clinical Practice Guideline: 'Use of Positive Airway Pressure Adjuncts to Bronchial Hygiene Therapy'," *Respiratory Care*, 1993; 38:516-521.

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